



In Memory of
Daniel Frederick Levy, M.D.
Class of 1919
Yale University
School of Medicine





DANIEL F. LEVY.

calo



INFANT-FEEDING

ITS PRINCIPLES AND PRACTICE

BY

F. L. WACHENHEIM, M.D.

ATTENDING PEDIATRIST, SYDENHAM HOSPITAL AND MOUNT SINAI DISPENSARY
NEW YORK CITY



LEA & FEBIGER
PHILADELPHIA AND NEW YORK

Entered according to the Act of Congress, in the year 1915, by $\texttt{LEA} \ \& \ \texttt{FEBIGER},$

in the Office of the Librarian of Congress. All rights reserved.

DEDICATED

то

MY FIRST TEACHER IN PEDIATRICS

DR. ABRAHAM JACOBI



PREFACE.

This manual is designed to supply the practitioner with a reference hand-book, of moderate size, in which he may find an exposition of the present theory and practice of infant feeding. The material here presented can only be secured by reading a vast journalistic literature, which is extremely conflicting in its statements and of little practical use, without extensive sifting.

In giving some five hundred literary references I have endeavored to select the articles that are most valuable and accessible, sometimes regardless of priority. These references do not, of course, include a tithe of the immense literature on this subject, but the reader will find most of the leading articles of the last decade duly noted.

F. L. W.

New York, 1915.



CONTENTS.

CHAPTER I.

Infantile Digestion and Metabolism.

Digestion; protein metabolism; fat metabolism; the infant stools; carbohydrate metabolism; salt metabolism; growth and weight; caloric requirement

17

CHAPTER II.

Breast-feeding.

Colostrum; composition'of human milk; milk secretion; capacity for nursing; contra-indications to nursing; physical and chemical tests; rules for breast-feeding; difficulties in breast-feeding; wet-nursing; preserved breast-milk.

48

CHAPTER III.

Cow's Milk.

Composition; analysis of cow's milk; the proteins; the fats; the carbohydrates; the salts; the ferments; the bactericidal property of milk; milk bacteria; milk infection; milk regulations; sterilization; pasteurization; condensed milk; desiccated milk; frozen milk; preserved milk; milk stations; whole-milk feeding; milk idiosyncrasy; milk of other animals

76

CHAPTER IV.

MILK MODIFICATION.

Simple dilution; removal of the casein, whey; removal of the fats, skim milk; removal of casein and fat; buttermilk; removal of whey, albumin milk, albumin-creammilk; addition of fat, top milk, Biedert's mixture, Meigs' mixture, Rotch's method (percentage feeding), Gärtner's fat milk, Székely milk, Niemann's modification; addition of protein and fat, casein-fat-milk, whey-modified milk

111

CHAPTER V.

MILK MODIFICATION (CONTINUED).

Addition of carbohydrates, malt, dextrin, starch; Jacobi's method, the author's modification, Pfaundler's variation; Keller's method; caloric feeding; the addition of alkalies; the addition of ferments, proteolytic, glycolytic; lactic acid fermentation; alcoholic fermentation; homogenized milk; baby foods .

144

CHAPTER VI.

OTHER INFANT FOODS.

Beef-juice; beef-extract; meat-broth; eggs; leguminous vegetables; carrots: hemp-soup; banana-meal; addition of water to diet .

174

CHAPTER VII.

ARTIFICIAL FEEDING.

The present situation; general recommendations; constipation; colic; vomiting; diarrhea; soap stools; feeding in the second half-year; mixed feeding; the feeding of premature infants

184

CHAPTER VIII.

DISORDERS OF DIGESTION.

Physical disorders: pyloric stenosis; pylorospasm; megacolon; singultus; rumination; digestive neuroses

206

CHAPTER IX.

DISORDERS OF DIGESTION (CONTINUED).

Alimentary disorders: protein indigestion; fat indigestion; carbohydrate indigestion; salt disturbances; Finkelstein's theory: disturbed equilibrium, dyspepsia, atrophy, intoxication

220

CONTENTS

CHAPTER X.

Infectious disorders: etiology, pathology, symptoms, prognosis, prophylaxis, treatment

243

CHAPTER XI.

DISORDERS OF METABOLISM.

The exudative diathesis; anemia; acidosis; the metabolic dermatoses: eczema, urticaria

267

CHAPTER XII.

RICKETS AND SCURVY.

Rickets: etiology, symptoms, diagnosis, prognosis, treatment; spasmophilia. Scurvy: etiology, sympto- 284 matology and diagnosis, treatment

CHAPTER XIII.

THE FEEDING OF OLDER INFANTS.

Weaning; feeding during the third half-year; feeding during the fourth half-year; feeding troubles

308

BIBLIOGRAPHY INDEX

321 333



INFANT-FEEDING.

CHAPTER L

INFANTILE DIGESTION AND METABOLISM.

Saliva is secreted at birth; diastase (amylase) is present in it and very active at birth; maltase and potassium sulfocyanid appear later (Ibrahim¹). The quantity of saliva is from 10 to 20 per cent. of the volume of the ingested food (Allaria¹). The act of sucking starts a free flow of saliva through the secretory salivary reflex (Ibrahim¹).

The mixture of saliva with the ingested milk facilitates the penetration of the rennin-casein curds that are formed in the stomach by the hydrochloric acid of the gastric juice (Allaria²). The diastase works best in a feebly acid medium, and therefore continues to act during the earlier period of gastric digestion so long as there is little or no free hydrochloric acid in the stomach contents (Chittenden and Griswold).

The act of sucking also starts the flow of gastric juice (Nothmann¹). Hydrochloric acid, pepsin, rennin, and lipase are present at birth even before any food is given (Hess¹). Free hydrochloric acid does not appear in the stomach contents until an hour or more

after a meal (Leo); its secretion is favored by alkalies, large doses of which interfere with the gastric digestive function, but is retarded by an excess of sodium chlorid, sugar, or fat in the ingesta, as well as by prolonged starvation. The total acidity is highest two or three hours after feeding, according as the infant is breast-fed or bottle-fed (Heiman¹). The rennin coagulates the milk-casein, but it is still doubtful how far the pepsin peptonizes the proteins. Davidsohn thinks that rennin and pepsin are identical.

Lipase was first found in the stomach by Volhard; in the infant stomach by Sedgwick; it splits only a small proportion of the ingested fats in the stomach, but continues active in the upper portion of the small intestine (Sedgwick and Schlutz).

Of organic acids, only lactic acid is occasionally found in the stomach of breast-fed infants; in bottle-fed infants the volatile fatty acids, especially butyric acid, are not infrequently present (Heubner¹).

High acidity favors closure of the pyloric orifice, but the stomach begins to empty itself about the time that free acid appears, that is, about an hour after the ingestion of breast-milk, and about two hours after artificial feeding (Czerny and Keller). The stomach is entirely emptied about an hour and a half later, but high fat percentages and excessive dilutions of cow's milk lengthen this period, whereas moderate dilutions, up to half and half, shorten it (Tobler and Bogen).

The stomach capacity of the normal infant has often been much underrated. The customary short

feeding-interval, which rarely permits the stomach to empty itself thoroughly, gives figures that are far too small. The following table, compiled from the data of a number of investigators, gives in ounces the total daily amount of food and the average quantity taken at each feeding. The figures refer to breastfed infants, nursed five times a day, at four-hour intervals, and were obtained by careful weighing immediately before and after feeding.

Age	One day.	Two days. oz.	Three days. oz.	Four days. oz.	Five days. oz.	Six days. oz.	One week. oz.
Per day	$\frac{2}{0.4}$	4	6	8	10	11	12
Per meal	0.4	0.8	1.2	1.6	2	2.2	2.4
Age	Two weeks.	Three weeks.	Four weeks. oz.	Five weeks. oz.	Six weeks. oz.	Seven weeks. oz.	Two mos. oz.
Per day	14	17	20	22	24	26	28
Per meal	2.8	3.4	4	4.4	4.8	5.2	5.6
Age	Three	Four	Five	Six	Seven	Eight	
	mos.	mos.	mos.	mos.	mos.	mos.	
	oz.	OZ.	oz.	oz.	oz.	oz.	
Per day	30	32	33	34	35	36	
Per meal	6.0	6.4	6.6	6.8	7.0	7.2	

The quantities given by the percentage method according to Ladd's¹ table (V, p. 129) are as follows:

Age	On wee		Two weeks.	Three weeks.	Four weeks.	Six weeks.
Per day . Per meal	10		oz. 15 1.5	oz. 18 2	oz. 20 2.5	oz. 24 3
Age	Two mos. oz.	Three mos. oz.	Four mos. oz.	Five mos. oz.	Six mos. oz.	Eight mos. oz.
Per day . Per meal	$\frac{24}{3}, \frac{5}{5}$	$\frac{28}{4}$	$\frac{27}{4.5}$	$\frac{33}{5.5}$	36 6	$\frac{42}{7}$

Ladd's figures for the stomach capacity are certainly too low for the early months of infancy, and his totals per day, from the eighth month onward, at present are considered excessive. Forty ounces per day should 20 I.

be the maximum amount of fluid given, and many foreign pediatrists set the limit at thirty-two ounces, or one quart.

The secretion of bile begins within twelve hours after birth, and rapidly becomes profuse, being fully established within a week or ten days. Failure of the organism to get rid of the secreted bile with equal rapidity is the probable cause of icterus neonatorum (Hess²). The long accepted theory of Quincke, which makes the presence and intensity of icterus neonatorum depend on the relative patency of the ductus venosus, has never been sufficiently substantiated by experiment, and is probably at the most a partial explanation.

All the pancreatic ferments are present, though scanty, in the newborn; their quantitative development is as follows: lipase increases rapidly in amount (Hess³), trypsin less rapidly, but more so than the diastatic ferment (Friedländer). The secretion of pancreatic juice is not suspended even in severe illness, but the lipase undergoes a marked decrease (Hess³). The secretion does not cease if the production of gastric juice is scanty, but, on the other hand, it is not promoted by the entrance of free hydrochloric acid into the duodenum (Hess¹). It should be remembered that trypsin and the pancreatic lipase are both secreted as zymogens, the former being activated by the enterokinase secreted by the small intestine, the latter by the bile (Uffenheimer).

The precision as to the facts just stated is due to the employment of the stomach tube and duodenal catheter. Below the duodenum our knowledge is necessarily less exact, not only with regard to the stages of digestion but also with respect to the sites of ferment activity. Besides the complementary action of the enterokinase, just mentioned, the ferment erepsin converts the previously formed protein derivatives into crystalloids of the amino-acid group, which appear to be absorbed directly (Folin), while maltase, invertase, and lactase exert their several specific actions on the carbohydrates. Diastatic action is feeble or absent in the intestinal secretion. Our information as to the minor details of digestion in the jejunum and ileum is still somewhat defective, because of the technical difficulties involved in this field of research.

We must note that the intestine is relatively long in infants, in proportion to the body length, and its musculature comparatively feeble; digestion is therefore relatively slow and thorough, but, on the other hand, more subject to derangement by such substances as are either directly toxic, or influence the peristaltic function. The excessive length and tortuousness of the sigmoid flexure tend to favor constipation in infants whose muscular system is underdeveloped.

Infants vomit with comparatively little effort; the food seems almost to overflow from an overloaded stomach. This phenomenon is certainly not due to remarkable vigor of the gastric muscularis, but is associated with the relatively feeble closure of the cardiac orifice in early life.

The secretion of hydrochloric acid keeps the stomach contents relatively germ-free, and the upper intestine is likewise nearly free from bacteria, because

of the antibacterial nature of the intestinal secretion. This safeguard largely fails in the presence of digestive disorders and during starvation (Moro¹); on the other hand, abnormal bacteria are rapidly eliminated by the healthy intestine, though the process may be accompanied with diarrhea (Jehle and Pincherle). Bacteria swarm in the large intestine, the species varying greatly according to the nature of the food and the condition of the digestive functions.

The passage of the ingesta through the gastrointestinal tract is normally accomplished in eighteen to twenty-two hours, according to tests with carmine, made by Triboulet, which require confirmation by other investigators. Bahrdt and Bamberg have shown that peristalsis is increased by a daily dose of 0.2 to 0.5 gram of acetic acid, less so by a similar dose of the other volatile fatty acids, and not at all by lactic acid. The acids, that are normally found in the digestive tract, do not therefore affect peristalsis perceptibly, under ordinary conditions.

PROTEIN METABOLISM.

We are in possession of quite a number of studies on the protein metabolism of infants. The earliest, made by Rubner and Heubner, are still among the best, and are wonderfully thorough; those of Keller are extremely valuable. Among more recent investigations, those of Hoobler are particularly useful, partly because they deal with rather high protein and fat feeding.

Hoobler's data for the ultimate disposition of the proteins, measured as nitrogen, are as follows:

Percentage passed in feces	3.9		
Percentage absorbed	96.1	as urea	45.4
Percentage passed in urine	63.7	as ammonia . as amino-acids	$\frac{9.5}{8.0}$
Percentage passed in urine and feces	67.6	as ammo-acids	0.8
Percentage retained	32.4	(as orcasinin	0,0

I have corrected Hoobler's figures slightly, to eliminate some obvious mathematical errors. Hoobler does not allow for the nitrogen derived from the metabolism of the intestine itself, whereby he would show the nitrogen loss through the feces to be still smaller.

If we turn to the amounts of protein and nitrogen, ingested by the normal infant we obtain the following figures:

	Early	Six	\mathbf{Ten}
	infancy.	months.	months.
	Gm.	Gm.	Gm.
Protein	10	25	40
Nitrogen	1.6	4	6.4
Nitrogen per kilo of weight	0.3	0.5	0.7

Early infancy is well represented by Keller's¹ data for an eleven-pound infant (5 kilos). Rubner and Heubner's subject weighed seventeen pounds (8 kilos); Hoobler's a trifle more. The first two seem to have been slightly underfed, if we are to judge by the daily weighings; the third was apparently somewhat overfed, but without showing any signs of digestive disturbance.

The essential accuracy of these researches seems to be shown by their substantial agreement; the three infants retained respectively 36, 26, and 32 per cent. of the ingested nitrogen. The daily observa-

tions, however, show very considerable fluctuations, especially with respect to the urinary ingredients, so that our present knowledge on this subject can be regarded only as approximate. The unavoidably short periods of observation, averaging seven days, have undoubtedly affected the value of the results materially; the chief obstacles have been, and probably will always be, the technical difficulties and the vast amount of labor involved in these and similar investigations.

Some points, indeed, appear fairly well settled. (1) Very little available nitrogen is lost in the feces of the normal infant; we shall see, however, that this applies solely to animal proteins, and that the absorption of vegetable proteins, for example, from cereals, is very much less complete. (2) We may, with Hoobler,1 assume with some confidence that about one-third of the ingested protein is assimilated, regardless of the quantity given. This figure, however, does not apply to colostrum feeding, as met with in the earliest period of breast-feeding. In this matter Birk¹ sustains the older reports of Michel, which gave twice as high a ratio of protein assimilation in the newborn. Birk! shows that this difference is intimately connected with colostrum feeding, because it does not occur in artificially fed newborn babies, who assimilate only about one-third of the ingested protein. A good résumé of the whole subject is given by Schwarz.

Frank and Schittenhelm have shown that the different milk proteins are absorbed about equally well, and similar utilization is made of the other animal proteins. Chittenden and Mendel claim that the absorption of vegetable proteins is about the same, but admit that they are thoroughly digested only when isolated; in their usual association with indigestible matter, such as cellulose, their absorption is imperfect. Milner and Atwater give a percentage of availability ranging from 72 to 88 per cent., the latter for fine wheat flour, whereas, as we have seen, from 96 to 98 per cent. of the animal proteins are digested.

There has been considerable discussion as to the presence of creatinin in the urine of healthy infants, but the finding of this substance, by Sedgwick,² in the liquor amnii, proves that it is a normal urinary constituent. High fat percentages in the milk do not affect protein absorption, but seem to increase the proportion of nitrogen excreted in the urine as ammonia (Hoobler¹). The excretion of nitrogen is increased by the administration of thyroid extract, but this is accomplished at the expense of the body proteins (Orgler¹). Some of the scanty excretion of nitrogen in the feees is not of alimentary origin, but is derived from the intestinal secretion and detritus (Orgler²), so that the absorption of the proteins is even better than the figures would indicate.

As shown in the preceding table the normal intake of protein per kilo of weight is approximately doubled during the first year, after which period it slowly declines. There is evidently a smaller requirement of nitrogenous matter in early infancy than later on, but the reason for this difference has not yet been given. We must, however, bear in mind that the

proteins, while indispensable to the growth and, more particularly, the muscular development of the body, play a minor part in the daily metabolism. An increase in weight cannot be obtained by ever so liberal a protein diet; even if it were possible to supply the theoretical caloric requirement in the form of nitrogen compounds, the body weight would nevertheless decline. This point will be more fully elucidated later on.

A few remarks on the purin bodies may not be amiss. It is well known that the kidneys, in the newborn, excrete a very large amount of uric acid, sufficient to form tube casts, and load the urine with an abundant red sediment. As this excess of pric acid is attended with a corresponding increase in the excretion of phosphates, we need not hesitate to refer it to the breaking up of nuclein, caused by an enormous postnatal destruction of leukocytes. As a matter of fact a marked polymorphonuclear leukocytosis appears at birth, which rapidly gives way to the normal infantile lymphocytosis. As the polymorphonuclear cells decline in number to the extent of about 20,000 to each cubic millimeter of blood, about 4,000,000,000 in all, the excretion of uric acid and phosphates rapidly rises to its maximum, speedily falling to the usual ratio, when the blood-count has reached its normal proportions, the entire process consuming about a week (Schloss and Crawford).

Aside from the occasion just discussed, the exerction of uric acid is relatively very low in infancy, because the baby's normal diet is particularly free from purin bodies, milk being quite exceptional in this respect.

FAT METABOLISM.

Of the ingested fat a little is split by the gastric lipase, more by the pancreatic lipase, still more is merely emulsified by the intestinal secretion. Neutral fat cannot be absorbed (Connstein); it must pass through the intestinal wall as either an emulsion or an alkaline soap, and is believed to do so chiefly as the former. Both fat derivatives are rapidly converted back into neutral fat, which is practically the only fatty compound found in the lymph stream, but the exact manner and place of this reversal is still obscure (Whitehead, Mendel²). In all, from 90 to 98 per cent. of the ingested fat are absorbed under the normal conditions of breast-feeding (Czerny and Keller), and this takes place almost exclusively in the small intestine, for absorption by the stomach is practically nil, and in the colon it amounts to very little.

Osborne and Mendel have shown that milk-fat contains certain ingredients essential to the growth of young animals that are absent in the ordinary fats; McCallum and Davis find the same properties in eggfat, and Mendel² finds them in cod-liver oil.

The Infant Stools.—The stools of the infant furnish the most available means of studying its fat metabolism; one of the best brief descriptions of the various types of intestinal evacuations is furnished by Morse.¹

Fat and its derivatives form about 50 per cent. of the dried feces in early infancy; as the infant grows older the ratio gradually falls to one-half and onefourth of this amount. In later infancy, neutral

fat and alkaline soaps each amount to about 10 per cent, of the total stool fat, the remainder consisting of varying proportions of free latty acids and soaps of the alkaline earths (Usuki). We must not forget that a moderate amount of free latty acid in the infant stool is normal (Escherich¹).

The normal breast-milk stool is semisolid, of a light yellow color, an acid but not fecal odor, and spreads out quite smoothly; signs of undigested fat should not be obvious, the visible evident presence of soft, yellowishwhite fat masses, commonly but inaccurately designated as fat-curds, is always on the borderline of the pathological, though subjective symptoms of indigestion may be lacking. The stools of bottle-fed infants are more than twice as voluminous (Rubner and Heubner). chiefly because they contain a much larger proportion of fat and fat derivatives; the odor is different, with a tendency to rancidity; and the occurrence of visible fatty particles is both more frequent and of more serious import. In addition, under certain conditions to be specified later, we observe true cheesy curds, hard, white, and more or less bean-shaped, which must be carefully distinguished from the so-called fat-curds.

Soap stools are found only in artificially-fed infants. They are pale or earthy yellow in color, of a firmer consistency than normal stools, and have been aptly compared to putty. They owe their appearance and greater solidity to the presence of large quantities of the insoluble soaps of calcium and magnesium. Soap stools are the characteristic feces of infants who suffer from poor fat absorption, without signs of acute or subacute indigestion.

All infant stools contain some free fat, but the socalled fat stools contain it in large amount. They are bright yellow, visibly oily, and leave a grease spot on linen and paper. They are invariably a sign of almost total failure even to emulsify, much less to digest, the food fats, and therefore a valuable sign of serious functional derangement; their bearings on infant dietetics will be more fully discussed later on.

Mucus is not normally present in the stools in sufficient amount to be macroscopically demonstrable; neither is it found in soap stools, for the formation of mucus tends to check saponification (Hecht). It is, therefore, when present in any quantity a trustworthy sign of serious digestive disturbance, and is practically found only in the presence or as a consequence of bacterial infection of the intestinal wall.

Talbot¹ recommends the following tests for the microscopic determination of the different fat derivatives in the feces. Carbol-fuchsin does not stain neutral fat, stains the fatty acids bright red, and the soaps dull red. A 5 per cent. alcoholic solution of Sudan III does not stain soaps, stains neutral fat red, fatty acid droplets red, fatty acid crystals unevenly. These tests, being merely qualitative, are of course of limited value for exact diagnosis. Cowie and Hubbard estimate the fats in the stools of infants by means of a modification of the Babcock method.

Green stools have recently become the subject of thorough investigation. Wernstedt's discovery of a

peroxid-splitting ferment in the feces has been utilized by Koeppe¹ in the study of this question. Koeppe¹ finds that the green color is due to three factors: first, the presence of a peroxid; secondly, the peroxid-splitting ferment; thirdly, a coloring matter turning green with nascent oxygen, probaly bilirubin. He conceives that this reaction may take place under conditions of perfectly normal digestion, but this hypothesis needs substantiation. It is better to consider green stools as symptoms of a pathologic digestive or metabolic process, possibly of very diverse significance. No good explanation has yet been offered of the relative frequency of this phenomenon in infancy.

During the first few days of life, instead of true feces, the infant passes the dark brownish-green substance known as meconium, which is not derived from the digestive processes, but consists of the fetal debris of the digestive tract and its accessory glands. The addition of true fecal matter to the meconium sets in after two to five, usually three or four days, and the evacuation of typical milk stools is fully established in from five to twenty, on the average ten days. The precise determination of this last date is sometimes difficult, as the feces may still be somewhat greenish-brown on the surface, and yet show the normal yellow color, when spread out (Southworth).

CARBOHYDRATE METABOLISM.

In considering the metabolism of the carbohydrates, we must distinguish between infants fed with the normal sugar of milk (lactose), and those fed with other carbohydrates, of which the digestion and assimilation are still perfectly possible within physiological limits.

As milk-sugar (lactose) cannot be absorbed as such, and as the milk-sugar splitting ferment, lactase, is secreted only by the small intestine, the metabolism of this carbohydrate does not begin until the duodenum has been passed. The ferment lactase is present from earliest infancy, for Nothmann² found it in a series of premature children; lactase splits lactose into galactose and glucose, both of which are absorbed, conveyed by the portal blood-stream to the liver, and there converted into and stored as glycogen.

Cane-sugar (saccharose), like most of the other disaccharids, is not absorbed as such, but must first be split by the invertase of the intestinal secretion into the two glucoses, dextrose, and levulose, which are readily absorbable. Maltose (malt-sugar) occupies an exceptional position among the disaccharids, in being partly absorbable as such. This is probably due to the fact that it can be split not only by the maltase of the digestive juices, but also by the same ferment being present and active in the circulating blood (Chittenden and Mendel).

To a certain extent, starch may also be assimilated by the normal infant. It must first be changed to dextrin by the diastase of the saliva, which we have shown to continue active during the earlier stages of gastric digestion, as well as by the same ferment secreted by the pancreas. The dextrin thus formed is still further changed into maltose by the same ferment, after which the further reduction, mentioned in the preceding paragraph, takes place. It will be seen that, although lactose is, strictly speaking, the only normal carbohydrate for the infant, a considerable number of allied substances is available for the same purposes.

These various carbohydrates, while capable of replacing one another quite well, as far as regards caloric value, are by no means exactly equivalent with respect to their relation to the digestive functions, even aside from the facts already related. Hartie, as well as others, notes that lactose particularly favors the growth of the bacteria that are most characteristic of the flora of the infant intestine, namely, the lactic acid bacilli. Nothing is better established than the fact that lactic acid fermentation, the acid being directly formed by bacterial action from a part of the ingested lactose, is essential to the maintenance of normal conditions in the intestine; it replaces the decomposition and putrefaction that take place in the intestinal contents of the adult. Skatol, indol. hydrogen sulfid, and similar substances are not found in the normal infant's intestine, and are always associated with more or less disturbance, when present under pathological conditions. Lactose also keeps down the formation of soaps in the intestinal contents (Freund¹); we shall see, in discussing the infant's mineral metabolism, that reduced exerction of soaps favors the retention of a greater proportion of the important element calcium, to the significance of which we shall

often have occasion to refer. The precise manner, in which the formation of calcium soaps is restricted, is still very far from clear, and cannot become so until we learn in which chemical combination the intestinal absorption of calcium takes place. In close relation to the last mentioned function, lactic acid is the physiological preventive of that bugbear of infant-feeding, namely, constipation, which is so difficult to avoid when other carbohydrates than lactose are given (Calvary¹); on the other hand, lactic acid does not, like some of the lower fatty acids, tend to promote peristalsis and cause diarrhea (Bahrdt and Bamberg).

Anticipating a little, we may mention that all cases, in which lactose may advantageously be replaced by other carbohydrates, are pathological, and without exception the result of unsuccessful attempts at artificial feeding; they will therefore be discussed under that head.

Dextrin, intermediate between sugar and starch, is physiologically nearer to the former; we shall have occasion to see that, under certain conditions, it may supplement sugar very advantageously. Given together with maltose, it materially delays the fermentation of the latter; Stolte¹ observes that the more complex the carbohydrate the longer fermentation is postponed.

As might be expected, starch is a relatively undesirable infant food, though it has played a very extensive role; whereas most babies take sugar about equally well, they differ very widely in their tolerance of starch; evidently there are great variations in the diastatic

function. Starch is usually given as a decoction of some cereal, the favorites being oatmeal, barley meal, and, in Germany, wheat flour. Most of the advantages afforded by cereal feeding are due to other factors than their starch content. The cereals favor bacterial growth far more than do the sugars; oatmeal distinctly fosters the formation of the volatile fatty acids, and consequently is decidedly laxative; wheat flour can hardly be said to affect peristalsis appreciably; barley flour lies in between the two (Klotz, Philips). Cereal feeding leads to a rapid loss of salts (Salge¹), a very undesirable feature, which is by no means compensated by the fact that the cereal carbohydrate, namely starch, is well utilized if given in moderate quantities (Pfersdorf and Stolte). We shall presently see that the maintenance of the body's salt-equilibrium is one of the essentials in successful infant feeding; on the other hand, since the absorption of lactose is practically perfect in the normal infant, there can only exceptionally be any advantage in substituting for it another carbohydrate.

Fats and carbohydrates can, to a certain extent, replace each other in the body economy, but this mutual compensation has certain well-defined limits. The normal food of the infant contains much more fat, in proportion to its weight, than is consumed by the adult; its carbohydrate intake is somewhat, but not decidedly lower. Fat therefore clearly plays an exceptionally important part in the metabolism of early life; it is not only stored under the skin and elsewhere as a fuel reserve, but it also acts as a conservator

of the body heat through its low conductivity. Sugar, on the other hand, is valuable almost solely as a body fuel, and is stored only to a small extent, chiefly in the liver, as glycogen. Proteins should not be expected to replace either the fats or the carbohydrates, though they may also supply body heat in an emergency; this function should, however, be kept down to a minimum whenever possible, and failure to do so is a fundamental dietetic error. When we go on to study the caloric requirements of the infant, we shall see that they are very much higher than in later life. The caloric value of fats is more than twice as great as that of the other foodstuffs; it is evident that the capacity to absorb fats is the basis of the infant's well-being. and that proteins and carbohydrates, to achieve similar results, even from the purely caloric point of view, must be given in quantities that are unmanageable because of their bulk, aside from other reasons.

METABOLISM OF THE SALTS.

According to Camerer and Söldner the body of the newborn infant contains 75 grams of salts, of which about three-fourths is calcium phosphate, and one-tenth each sodium and potassium chlorids; magnesium salts amount to somewhat more, and iron compounds to somewhat less than 1 per cent. each. Human milk contains far more salts, with the exception of iron, than are required by the infant, but the supply of iron is only just sufficient. We shall see that the circumstance that cow's milk contains about four

times as much mineral matter as human milk, is of no advantage whatever to the artificially-fed baby; incidentally, the supply of iron in cow's milk is very inadequate.

Sodium chlorid is not absorbed through the intestinal wall solely in conformity with the local osmotic conditions, and is retained in the organism in exactly the amount required to keep up the physiological concentration. Excessive feeding with sodium chlorid is very apt, in the infant, to lead to excessive water retention (Freund²); E. Schloss¹ has shown that the ingestion of 0.5 to 0.75 gram of sodium chlorid increases the body weight from 60 to 120 grams, the gain consisting wholly of water, which is added practically in the physiological ratio. In other words, an excess of salt causes an accumulation of water in the blood and other tissues in about the proportion of 1 to 120, and this takes place the more readily because sodium chlorid is excreted only very slowly by the infant kidneys, and hardly at all in the feces.

Potassium chlorid is far less apt to cause disturbance of the water equilibrium. E. Schloss¹ has demonstrated the relatively rapid elimination of potassium chlorid by the kidneys. Theoretically, potassium salts are more toxic than sodium salts; clinically, the more rapid elimination of the former more than compensates for their greater toxicity.

Freund² has shown that the retention of sodium chlorid and water is not accompanied by the laying on of fat; these two processes are opposed to each other, and cannot take place simultaneously. In accord with this are the findings of Steinitz, who shows that high fat feeding tends to lower the assimilation of the alkalies, with an increased output of ammonia in the urine.

The absorption and assimilation of calcium present a very complicated problem. The absorption of calcium from cow's milk, although that element is five times as abundant as in human milk, is relatively not so good, though quantitatively considerably greater; furthermore, the loss of calcium through the feces, the main route of elimination, is so many times greater in artificial feeding, that the net assimilation is less than from human milk. The loss occurs chiefly in the form of insoluble soaps, formed through the binding of the calcium by the undigested cow-milk fats in the lower intestine. Hoobler² seemed to secure better calcium retention with high fat feeding, than when he gave mixtures that were low in fat. There is reason to believe that this experience will be verified by other observers, but as a practical routine measure for promoting calcium retention it is open to objections that will be discussed more fully under the head of artificial feeding. Let it suffice here to say that this measure is replete with danger, especially in early infancy, as past experience has abundantly shown. The recent efforts of Stolte² and others, to revive interest in high fat feeding, refer to fat percentages that, not long ago, would have been regarded as inadequate.

Iron is barely sufficient in human milk, to meet the infant's needs; in cow's milk there is a marked deficiency, the importance of which will be considered later. The conditions of absorption and assimilation of iron are very similar to those of calcium. The chief need of iron is of course to maintain the hemoglobin content of the blood.

The metabolism of magnesium is so closely allied to that of calcium that a separate discussion is hardly necessary.

We have already stated that calcium phosphate constitutes about three-fourths of the mineral matter of the infant's body: the metabolism of phosphorus therefore is of exceedingly great importance and interest. First, its absorption and assimilation, as might be expected, run closely parallel to those of calcium (Schabad¹). Secondly, its excretion, which is effected chiefly in the urine, is in inverse ratio to the loss of fat in the feces; in other words, if the calcium is not bound to fatty acids in the intestine, it has a better opportunity to combine with the ingested phosphorus, and vice versa (J. and W. Cronheim). This would make it appear that the retention of phosphorus is less easily interfered with than that of calcium, consequently the promotion of calcium retention is the crux of the mineral feeding problem. Thirdly, we can improve the assimilation of calcium by giving phosphorus (Schabad¹), but this element must be given either uncombined, or in certain protein combinations such as casein. Berg has shown that phosphorus, in the forms of calcium or other phosphates, as well as glyccrophosphates, hypophosphites, phytin, and lecithin, is quite useless for this purpose. I need hardly add that the chief function of the absorbed calcium and phosphorus is to promote the bone formation of the growing infant, though considerable amounts are also needed for the development of other tissues, especially the central nervous system.

Knox and Tracy have shown that the excretion of phosphorus in the urine is greater in artificial than in breast-feeding, but they admit that the nature and severity of the nutritional derangement, thus indicated, cannot be estimated with any degree of accuracy.

Sulfur forms less than 1 per cent. of the mineral substance of the body and, until Freund³ began his investigations, had been studied very little. Hoobler² finds an absorption of 92 per cent. and an assimilation of about 30 per cent. under favorable conditions. Elimination takes place mostly through the urine. As it is a protein constituent, its functions must be of the greatest importance; our knowledge of these, however, is so far very inconsiderable.

The water metabolism of the infant is relatively simple; we have seen that it is in a direct relation to the absorption of the alkaline chlorids. More water is excreted than absorbed, for a considerable quantity is produced in the combustion of the proteins, fats, and carbohydrates. A certain minimum of water must be ingested, to maintain the normal salt metabolism; Meyer¹ sets this minimum at 15 per cent. of the body weight per day, O. and W. Heubner consider 12.5 per cent. sufficient and most of the feeding tables provide for about 10 per cent., which is probably enough under ordinary conditions. Much, quite naturally,

40

depends on such matters as the increased respiratory loss of water in a very active infant, or the increased transpiration through the skin in hot weather, so that we cannot set an arbitrary standard. Both Meyer¹ and Niemann¹ have observed a tendency of the body weight to become stationary or even decline, if insufficient water is given.

The gas metabolism of the infant need not detain us long; it differs from that of later life chiefly in its greater activity; both the intake of oxygen and the output of carbon dioxid are relatively large. The latter interests us especially, as being an index of the caloric energy of the body, a subject that will be taken up presently; otherwise, it presents no features that are in any way peculiar to early life.

GROWTH AND WEIGHT.

The newborn infant measures about 52 cm. (20.5 inches) in length, attaining about 61 cm. (24 inches) at three months of age, 67 cm. (26.5 inches) at six months, 74 cm. (29 inches) at one year, and 82 cm. (32 inches) at two years. The normal weight of infants is given in the accompanying table.

Breast-fed babies gain more regularly than bottlefed infants: the latter are more apt to present a zig-zag line on the weight-chart, to fall behind during the first few months, and to gain rapidly toward the end of the first year. The reasons for these deviations from the normal will be given later.

		v	Weight.		
Age.		Kilos.	Pounds.		
At birth		3.3	$7\frac{1}{4}$		
3 days		3.1	$6\frac{3}{4}$		
1 week		3.2	7		
2 weeks		3.4	$7\frac{1}{2}$		
3 weeks		3.6	8		
1 month		3.8	81/2		
2 months		4.7	$10\frac{1}{4}$		
3 months		5.5	12		
4 months		6.2	$13\frac{1}{2}$		
5 months		6.7	$14\frac{3}{4}$		
6 months		7.1	$15\frac{1}{2}$		
7 months		7.4	$16\frac{1}{4}$		
8 months		7.7	17		
9 months		8.0	$17\frac{1}{2}$		
10 months		8.3	184		
11 months		8.6	19		
12 months		9.0	20		
15 months		9.7	$21\frac{1}{2}$		
18 months		10.4	23		
24 months		11-8	26		

Even in successfully breast-fed infants, there is an initial loss of weight during the first two to seven—average three and a half days, amounting to about half a pound; the loss is not regained until the fifth to the twenty-second day—average ten days (Southworth¹). Many other authors, especially such as refer to infants in institutions, give much more unfavorable figures, but Southworth's¹ data are quite applicable to the children of well-to-do families in private practice. In bottle-fed infants, the initial loss is apt to be greater, and the recovery slower, so that it often takes a month or more to regain the original weight, even with the best of management.

The causes of the early loss of weight have been the subject of much speculation, but may now be regarded as quite definitely settled. In the first place, 42

the infant makes little effort to obtain food during the first, and sometimes also the following day (Aronstamm). Secondly, along with the deficit in the food intake, there is at the same time a shortage in the water intake; we have just seen that this is sure to involve some loss of weight. Townsend sets down a loss of $3\frac{1}{2}$ ounces for meconium, and $1\frac{1}{2}$ ounces for the vernix caseosa, both items possibly overrated. but still not to be entirely ignored. It is not quite accurate of Birk² to regard the newborn infant's balance as adequate, in spite of the decline in weight; disregarding the meconium, as composed chiefly of detritus, there is no question as to the loss of nitrogen and salts in the urine, and of carbon in the expired. air, the salts being drawn off with the excreted water. and the nitrogen and carbon derived from the disintegration of a certain amount of body protein, fat and carbohydrate. In accord with these facts, Cramer¹ regards the early loss of weight wholly, or at least partially, avoidable by early artificial feeding; he recognizes, however, the disadvantages and even dangers of this procedure, so that it hardly ranks as a practical measure. At this point, we may take note of what Holt has aptly designated as inanition fever. occurring in infants during the period of declining weight; he checks this rise of temperature promptly, by giving a few ounces of water in the course of the day. This simple procedure may very well be adopted, both to prevent inanition fever, and to keep the normal loss of weight within moderate limits; v. Reuss recommends it as a routine method.

While too much stress cannot be laid on the body weight as a test of the successful feeding of an infant, the above discussions will have been wasted, if we follow this standard blindly. We shall have ample opportunities to study its fallacies, when we come to the consideration of artificial feeding; for the present it will suffice to state that mere regard to the body weight, without allowance for the water and salt equilibrium, is a fruitful source of error in judging the progress of the individual infant.

CALORIC REQUIREMENT.

The study of the caloric requirement of the infant has, for many years past, engaged a large portion of the time and labor of pediatrists; the essential facts have, however, been understood for some time, and comparatively little of practical value has been added by recent investigations.

Let us begin with a brief sketch of the principles involved, and a definition of the terms employed. The sum of the work, performed by the body in twenty-four hours, is designated as the daily energy output; this is measured in large calories, one calorie being the quantity of energy required to heat one kilogram of water 1° C. The energy output includes two factors: (1) the functional metabolism of the body, which is roughly proportionate to its bulk, and (2) the maintenance of the body heat, which is proportionate to its radiating surface. The former varies enormously, according to the infant's muscular activity, mere

screaming raising it 50 per cent. (Schlossmann and Murschhauser). The latter varies chiefly according to the surrounding temperature and humidity, as modified by the clothing; it may be reduced to zero at the indifferent temperature—for infants 80° to 95° F., varying with the humidity, and higher in newborn than in older infants—or may even become negative, with symptoms of heat retention, namely, thermic fever (heat-stroke), in very hot weather, or in the incubator. In a cool room, or out of doors in ordinary weather, the radiation of heat becomes an important item, since the head, at least, is always more or less exposed, and protection by clothing cannot be made perfect. The loss of heat by radiation is relatively great in infancy, owing to the proportionately large body surface.

The total number of calories in the daily energy output, divided by the weight in kilograms, gives the figure called by Rubner the energy quotient. This should include the storage of energy, involved in the infant's growth, amounting to about 20 calories in early infancy, and 10 toward the end of the first year. The energy quotient is best stated as the intake, as more easily calculated, and the infant's weight regarded as the test of whether the output is larger or smaller.

Allowing for such imperfect absorption and metabolism, as lie within physiological limits, the net caloric value of the different foodstuffs, in large calorics, is as follows:

 Proteins
 4 calories per gram. 120 per ounce

 Fats...
 9 " " 270 "

 Carbohydrates.
 4 " " 120 "

One-half calorie should be deducted for vegetable protein, and one for the fat of cow's milk, if we desire real accuracy; this is not usually done, so that the caloric intake, in artificial feeding, is apt to be somewhat overestimated.

The salts—estimated as ash in analysis—have no caloric value, and the same is true of the ingested water.

Caloric estimation is not quite so simple as the above data would suggest, and the calculations of various observers differ so widely as to create some doubt as to their real value. I give a few illustrative examples, all expressed as energy quotients.

	First three months.	Second three months.	Third three months.	Fourth three months.
$\mathrm{Beck^1}$	107	91	83	69
Finkelstein ¹	100	95		80
Ladd ²	116	126	125	120
Boissonas	170	140		110

As a further contrast we may note 63 calories, obtained by Schlossmann and Murschhauser in a tenpound infant, 55 to 82 by Calvary² in young infants, and 70 by Budin and Planchon. Schlossmann and Murschhauser admit, however, that their figure applies to a very quiet baby, and that a restless or crying child gives a larger quotient. Of interest is the observation, first made by Cramer³ and Aronstamm, that the energy quotient during the first few days of life rarely exceeds 60; Cramer³ attributes this low figure to water retention, which explanation is far from satisfactory, as we have seen that there is a considerable loss of water at this period. To me the theory of Feer¹

seems much more reasonable; he thinks that the energy quotient varies according to the food-supply, and that the body tissues are economized when this is scanty: furthermore, at this time, instead of allowing 20 calories for the gain in weight, we may subtract an equal amount to represent the physiological loss; in this manner the low figures, in the first days after birth, seem readily explicable.

We do well to admit that all the work in this field has dealt purely with the theory of nutrition and that its arguments have been essentially a priori; the actual requirement of the infant is uncertain, variable, and probably only vaguely determinable; the idea that definite metabolic and nutritional results can be obtained from a definite caloric food-estimation has hitherto been, and will probably remain chimerical. We shall still be compelled to fit the amount of food to the physical requirements of the individual infant, and not expect to obtain definite results from a fixed quantum of ingesta; it is indeed doubtful if caloric investigations have been of great practical value in determining methods of infant-feeding. Even simple mechanical devices, made to resemble one another with mathematical precision, will not give equal results from equal supplies of energy; still less can such exactness be expected from the complicated and imperfectly understood mechanism of the human body.

It cannot be said that investigators have been unaware of these difficulties. One method of correction is to calculate the energy quotient with reference to

the surface of the body instead of its weight; we have already seen that both points of view are almost equally important. This plan was adopted by some of the earliest observers, who laboriously measured the body surface with paper strips; it is evident that this procedure is too delicate and difficult for every-day use, and therefore Meeh, Lissaucr, and Howland and Dana have endeavored to draw up simple formulas, by which the body area can be deduced from the length and weight. The results of these efforts have been very varying, and the possible fallacies are only too obvious: a ratio between surface and weight is gratuitously assumed to exist, without any real proof that such is the case. It is therefore evident that this modification hardly represents an advance; it is merely another unsuccessful attempt to reduce to simple figures a matter that is not thus reducible. The energy output and the energy quotient must be recognized as variable individual characteristics, to be considered chiefly as an additional check on the feeding methods, that will be described in detail in the following chapters.

CHAPTER II.

BREAST-FEEDING.

Human milk, strictly speaking, the mother's milk, is the infant's normal food during the greater part of the first year. It is therefore desirable to give as complete an account, as possible, of human milk and its relation to the infant organism, first, because the majority of babies are breast-fed, and secondly, because breast-feeding is the standard, to which we seek to adapt the various methods of artificial feeding.

COLOSTRUM.

The earliest secretion of the mother's breast, called colostrum, differs materially from true milk; its average composition is as follows (Camerer and Söldner):

Water			87.9 pc	er cent.
Proteins			 3.1	4.6
Fats.			3.3	F 6
Milk-sugar			5.3	1.6
Salts .			0.4	4.6

Colostrum is light yellow in color and decidedly alkaline, richer than milk in whey-proteins and salts, especially globulin and phosphorus, poorer in casein and sugar. The proteins may exceed 6 per cent. on the first day, and the caloric value 1000 per liter (Langstein, Rott and Edelstein). Microscopically the characteristic elements are the so-called colostrum corpuscles, which are cellular bodies of several times the diameter of leukocytes; there is present, besides, a large number of leukocytes. A high proportion of polymorphonuclear leukocytes (80 to 90 per cent.) gives promise of an ample secretion of milk later (Zuckerkandl).

After a few days a change begins to take place in the composition of colostrum, as it is gradually replaced by true milk; this process is usually complete by the twelfth day, but sometimes takes a month (Steele). Occasionally we note a partial reversion to colostrum characteristics, indicated by a reappearance of colostrum corpuscles; this may be the result of psychical shock, returned menses, supervening pregnancy, and rarely other causes. It is usually only temporary, so that withdrawal of the infant from the breast is rarely called for (Spolverini¹).

It has been determined, by hemolytic tests, that the colostrum proteins are, at least in part, derived from the blood (Bauer¹), whereas the milk proteins are entirely the product of the mammary glands. The hemolytic and bactericidal powers of the colostrum are, however, inconsiderable (Kolff and Noeggerath). The fat and sugar of colostrum are identical in kind with those of milk (Engel and Bode), but there is an excess of oleic acid in the former. The excess of salts is possibly responsible for its slightly laxative action, but this point is by no means settled.

COMPOSITION OF HUMAN MILK.

True human milk consists of a serous fluid, holding in suspension the fatty milk-globules, and in solution proteins, milk-sugar, salts, extractives, and gases; cellular elements are scanty in normal milk. Its chemical composition is very variable, and ranges about as follows:

Water	87.0 to	88.5	per cent.
Casein .	1.1 to	1.4	4.4
Lactalbumin and lactoglobulin	0.4 to	0.6	4.4
Fats	3.5 to	4.0	**
Milk-sugar	6.5 to	7.0	
Salts	0 2		4.6

Of the salts, potash amounts to about 7 cgm. per liter, phosphoric acid 4, lime and chlorin each 3, soda 2 cgm., and oxid of iron 2 mgm. The caloric value of human milk ranges between 640 and 720 calories per liter or quart, 19 and 22 per ounce.

Not only does the milk of different women vary greatly, but the same mother may yield very different milk on different days, and this without reference to the period of lactation (Sharples and Darling). Even the milk from the two breasts may differ, an important thing to remember in raising twins, who should be put to both breasts in rotation (Zappert and Jolles). The secretion is apt to be more abundant in the morning, than later in the day, probably because of the accumulation of milk overnight.

The proportion of proteins and fats is relatively low in primiparæ (Sharples and Darling). The fats are the most variable constituents of the milk;

compared to cow's milk, there is a relatively high percentage of oleates and palmitates, and a low proportion of stearates and the volatile fatty acids. The total amount of fat is low in the morning, and at the commencement of nursing, high in the evening, and as the breast is nearly emptied (Engel, Forest); the proportion of fat may vary from almost zero to more than 13 per cent. (Engel).

The proportion of sugar is comparatively constant, but somewhat higher in primiparæ (Sharples and Darling). It may be as low as 4 or as high as 11 per cent. (Schlossmann²), but these extremes are exceptional.

As to the mineral constituents of human milk, we may note that the calcium declines in amount toward the end of nursing (Hunaeus), and is also reduced by an insufficient intake of lime in the food (Dibbelt). It cannot, however, be pushed above the normal amount by an increase of lime salts in the mother's diet; it is, furthermore, subject to great variations and fluctuations, and declines in the later months of lactation (Schabad²). The proportion of iron runs nearly parallel to that of calcium, the percentage may, however, be increased by feeding the mother with iron (Bahrdt and Edelstein).

The percentage of phosphoric acid is in proportion to that of casein; it is more closely attached to that protein in human milk than in cow's milk, and occurs in a larger amount as lecithin (Siegfried). The importance of the last-named substance has been rated very high, because it has been regarded as a valuable source of phosphorus for the infant; since, however, Berg has shown that lecithin-feeding is ineffective for the purpose of making up a phosphorus deficiency, its value has been rendered somewhat problematical.

The other mineral constituents of human milk present nothing characteristic, but one of the most important points of all must, on no account, be omitted, namely, that human milk contains only about one-fourth as much mineral matter as cow's milk. We need here merely take note of this fact; it can be discussed more profitably when we consider the composition of cow's milk and its utilization as an infant food.

The ferments in human milk have been studied with considerable minuteness. Moro² finds that human milk exceeds cow's milk in hydrolytic and proteolytic ferment, as well as oxydase, lipase, and diastase (amylase), but he disagrees with the many authors who insist on the great importance of these ferments to the infant. Friedjung and Hecht have shown that babies thrive about as well when the ferments are scanty, as when they are abundant. Spolverini2 is one of those who think the ferments necessary; he shows how we can increase the proteolytic ferment and the diastase in the milk, by giving the mother pancreatin and diastase. We must add that Zaitschek could not find the proteolytic ferment, though he recognized the abundance of diastase. The absence of a glycolytic ferment is generally conceded.

Kolff and Noeggerath claim that human milk possesses no immunizing, hemolytic, or bactericidal

powers of any consequence; in view of the presence of the above named ferments, it is, however, difficult to deny it a slight restraining influence on bacterial growth.

MILK SECRETION.

Various methods of promoting the secretion of milk have been tried. Unquestionably, the best way of all is the act of nursing (Laisney); putting a robust baby to the breast may develop an ample flow of milk for the benefit of a puny infant whose powers of suction are feeble (Wile). The nursing of twins usually results in an increased supply of milk, quite sufficient to meet the double demand (Laurentius). In certain cases we may successfully promote lactation by means of the breast-pump (Helbich). We cannot, on the other hand, contrary to the popular belief, increase the secretion by overfeeding the mother, though a very scanty food-supply, as, for example, in a strenuous reduction cure, will diminish the flow (Schkarin, Keller, Malagodi, Engel¹), and the general physical deterioration called hospitalism has the same unfavorable effect (Genersich). Jaschke¹ increased the milk-supply by means of local hyperemia, obtained by Bier's method, and Nolf claims to have had good results from the hypodermic injection of about 10 c.c. of milk.

Zlocisti and a number of other authors think that cotton-seed is of value as a galactagogue. Ott and Scott find an increase in the flow of milk, following the administration of extracts of the pituitary, pineal and thymus glands, and of the corpus luteum. On

the question of malt, as a galactagogue, opinions are divided; beer has been used for this purpose from time immemorial, but this beverage, in any great quantity, is decidedly objectionable, because some of the contained alcohol is apt to pass into the milk. Whether the malt-extracts have any galactagogue action is still doubtful; the results from certain proprietary malt-protein compounds, such as malt-tropon, are very conflicting (Liepmann, Gewin). A great many French and Italian observers recommend aromatic substances, especially anise, but their reports are far from convincing. In short, the mechanical stimulus of suction seems still to be the most effective and only reliable method of increasing the secretion of the mammary glands.

It is well known that the breasts of the newborn infant begin to secrete milk in the first week of life, continuing to do so for some weeks or even months. This is probably due to the passage through the placenta of the hormones that activate the maternal mammary gland, the same biochemical process thus taking place simultaneously in both mother and child. Accordingly, Basch believes that the degree of activity of the infant's breasts may be regarded as a fair guide to the prospective milk production of the mother.

In conclusion, let us remember that any stimulation of the secretion of milk is finally limited by the amount of mammary tissue. Thiemich observes that the usual maximum is from a liter to a liter and a half per day, though two and a half liters may be secreted in exceptional instances.

For a good account of our present information on the subject of human milk, see Talbot⁴, who quotes from about 150 articles.

CAPACITY FOR NURSING.

How many women can nurse their babies successfully for at least six months? This question is not to be answered by statistics of the actual prevalence of breast-feeding, for, aside from certain possible but doubtful racial peculiarities, there is a host of social factors that interfere with the only physiological method of rearing infants. Davis' report for the situation among the poorer classes of Boston is of interest; he finds that of Italian mothers 86 per cent. nurse their babies, of Russians 82 per cent., of Irish 77, of native Americans 64, and of Canadians only 55 per cent. This shows a decline, partly in consequence of an improvement in social status, but more as the result of what might be called racial habit and certain complicated economic factors. At first sight, breastfeeding would seem more economical than bottlefeeding, but under our present labor system, the former interferes greatly with the mother's chance of earning a livelihood, where this is rendered necessary by the family circumstances. These two economic influences work in direct antagonism to each other, but in general it will be found that in communities where the wife stays at home the ratio of breast-feeding is high, whereas shop and factory life, on the part of the married women, lowers the ratio greatly, sometimes almost to zero. In the opinion of most hospital and dispensary observers the vast majority of mothers are physically able to nurse their babies. Jaschke² estimates that 98 per cent. can nurse for a brief period and 91 per cent. for several months. Schwarz² gives the following figures, based on a large material on the East Side of Manhattan, New York: 97 per cent. for one month, 91 for two months, 88 for three months, gradually declining to 77 for six, and 68 per cent. for ten months. Such favorable statistics can, however, be obtained only with the exercise of great perseverance in individual cases, when due attention is also given to social conditions. Reuben finds that the milk of many mothers deteriorates, or fails altogether, after the seventh or eighth month.

Stuhl briefly summarizes the organic causes of difficult nursing under three heads: (1) deficient development of the mammary glands; (2) malformation of the nipples: (3) a combination of both factors. Breasts approaching the virginal type are likely to furnish very little milk; a voluminous, pear-shaped, and somewhat pendulous gland is usually the most efficient; but the very pendulous, flat gland, seen in many middle-aged multiparæ, is less adequate. The erect nipple should be fully 1.5 cm. in length and about 1 cm. in diameter; smaller nipples are more difficult for the infant to take hold of, and some women have practically none at all. Stuhl thinks that in most cases the mentioned obstacles are only relative, and may be overcome by judicious management and patience, employing the breast-pump in extreme cases; many infants can thus be supplied with their normal food for a month or two, and even a short period of feeding with breast-milk represents a material gain.

CONTRA-INDICATIONS TO NURSING.

These may be divided into local and general contraindications, the former including local diseases of the breast itself, which are usually only unilateral, and therefore not an absolute bar to nursing.

If the nipples are carefully kept clean and dry during the latter months of pregnancy, they are not likely to give trouble afterward; if they are neglected, fissures are apt to occur; these are not so often of traumatic origin, as a manifestation of local eczema, due to lack of cleanliness, or maceration by the colostrum. One source of trouble is the old custom of frequent attempts at nursing on the first and second days after delivery. Some physicians encourage nursing from fissured nipples, if these are not painful; in my opinion the risk of infection is too great to permit this. It is safer to keep the child from the affected breast, remove the milk with the breast-pump under aseptic precautions, and apply silver nitrate and aseptic gauze locally until the fissures are entirely healed.

Mere caking of the breast is no contra-indication; in fact, vigorous suction is the best preventive of this condition, which is always serious, for combined with fissured nipples it is the usual precursor of mastitis. In mastitis, of course, nursing is absolutely contra-indicated in the interests of both parties.

The withdrawal of the infant, under the above conditions, involves the undesirable likelihood of the breast drying up, with the consequent deprivation of the infant of its natural food. Sometimes, when merely fissures are present, this unpleasant result may be avoided by using the breast-pump; in the case of mastitis, however, the gland is quite certain to cease functionating in a very short time.

Affections of the infant's mouth contra-indicate nursing only under very exceptional conditions; breast-feeding is the best preventive of infantile stomatitis and other local lesions.

Among general contra-indications, active maternal tuberculosis ranks first; this consideration comes up very frequently and the contra-indication is absolute. The drain of nursing is almost sure to hasten the progress of the disease, and there is great danger of infecting the baby, which is subject to an excessive amount of exposure in the intimate act of nursing. It must be remembered that the susceptibility to tuberculosis at this age is extreme. As to latent tuberculosis of the mother, opinions are not quite unanimous, but prohibition is advisable in these cases also. It is different in the case of a history of healed tuberculosis, especially of the bones or glands, or a successfully operated affection of the genito-urinary tract; here nursing is probably quite safe. Presumably healed pulmonary tuberculosis must, however, be regarded differently, as we can never be quite certain if this lesion is entirely cured, here also, I would interdict nursing.

Syphilis presents a many-sided problem. If the mother's luctic manifestations date back farther than a few weeks the infant may be assumed infected. and breast-feeding usually affords the only means of saving its life, for artificially-fed syphilitic babies do badly. If the mother's affection, as rarely happens, is so recent that the child may have escaped, it may be best to feed the child artificially and take no chance of its contracting syphilis; in these cases we must exercise our keenest judgment. If the child becomes accidentally infected after it is born, say at circumcision, nursing is absolutely contra-indicated. We have at present, in the Wassermann test, a reliable means of settling doubtful cases. I should like, however, to call attention to a recent paper by Lucas, in which he shows that it is not necessary for both mother and child to react positively, and that both, indeed, may react negatively, the father only presenting a positive reaction. Here, too, there is abundant occasion for the exercise of judgment, when the clinical signs of lues are not manifest.

The role of nephritis as a contra-indication to nursing has been the subject of considerable debate. In the case of recent eclamptic or uremic attacks there has been comparatively little controversy, and Frost has shown pretty conclusively that under these conditions the milk is toxic. Goodall goes further and forbids nursing even with mild nephritis, regarding mere albuminuria as a contra-indication; in this he encounters the opposition of, more particularly, the Italian observers, among whom Spolverini³ and

Francioni advise against nursing, only in the presence of severe symptoms, disregarding albuminuria alone. On the other hand, Mori and Concetti¹ are not quite convinced of the safety of this position. The subject is evidently one that must be considered according to individual circumstances; there certainly are some mothers, suffering from a mild type of nephritis, who can nurse their babies; in these cases it should not be difficult to decide whether or not the infant is doing well at the breast.

A well-compensated valvular heart lesion is certainly not a bar to nursing, but the slightest decompensation interdicts it absolutely; the attending physician should rarely be in doubt as to his decision in the individual case. Diseases of the nervous system are to be regarded The unfortunate child of an epileptic mother is surely sufficiently burdened, without submitting it to the risk of physical injury, that maternal nursing would involve; with regard to insanity the same conditions of course hold true. As to hysteria and neuroses of the milder types, the hereditary burden of the infant is not increased by taking its mother's milk; in these cases nursing is usually advisable. Sometimes, however, such a woman is temperamentally unfitted to take eare of a child, and in that event the physician must act accordingly.

In wasting diseases, such as exophthalmic goitre, pernicious anemia, and carcinoma, the supply of breast-milk is usually so deficient that the question of nursing hardly comes up; when it does arise an adverse decision is imperative in the interests of both mother and child.

For obvious reasons the acute infectious diseases form a contra-indication to nursing, but as to other acute febrile conditions there is some diversity of opinion. In general it is best to suspend or discontinue nursing when the mother's temperature exceeds 101° or 102° F., as, under these conditions, the milk is more than likely to possess toxic qualities and disagree with the infant. Subfebrile temperatures must be considered according to the individual case; the objections to artificial feeding must be carefully weighed against the possibly trifling risk to the baby if it be kept at the breast; in later infancy, conditions are relatively favorable for passing on to bottle-feeding, whereas in the earlier months this is to be avoided whenever possible.

Though the return of menstruation, during lactation, seems to have an unfavorable effect on the quality of the milk, it is, nevertheless, advisable not to discontinue nursing if the infant seems to thrive. According to Rotch,⁵ there is a loss of about 1.5 per cent of fat, and an equal gain of protein in the milk, during menstruation; Bamberg, on the other hand, claims that menstruation does not alter the chemical composition of the milk. Should pregnancy supervene the milk often ceases to flow, so that nursing ceases automatically; should, however, the supply of milk continue the same rule applies as for returned menstruation; some babies of pregnant mothers do very well at the breast, especially during the earlier period of gestation.

Of all the reasons given for discontinuing nursing

the most frequent is the plea that the milk lacks the proper nutritive value, or is insufficient in quantity. This argument should never be even considered as a bar to nursing, unless the child evidently fails to gain in weight for several weeks, and, in that event also, we should proceed cautiously. Mother's milk, that is well below the standard in quality, is nevertheless, as a rule, better for the infant than cow's milk, at least during the first few months. The physician should not let himself be misled, by a single analysis, to consider the milk inadequate; as a matter of fact, some babies get along fairly well on human milk which, on analysis, seems absurdly insufficient in nourishing qualities. This is, after all, not so remarkable, since the deficiency is usually in the fats, the least essential of the organic constituents; these may, as we have seen, be largely compensated by an adequate supply of milk-sugar, which hardly ever fails to be present in sufficient quantity. Quantitative insufficiency of the milk, as a whole, is a more serious matter, and usually ends, within a few weeks, in the unavoidable abandonment of breast-feeding; our statistics have, however. shown, that this unpleasant eventuality is not so much to be apprehended in the earlier months of lactation. as is generally believed both by physicians and the laity; later on, if rendered necessary, the change to bottle-feeding is a relatively harmless procedure.

The milk is regarded as flowing freely, if it exudes in numerous drops, when the breast is grasped with the hand, and pressed gently but firmly; after lactation is well established, and the supply is ample, the milk fairly squirts from the ducts. A drop of the milk, seen by reflected light, should be of a delicate bluish-white color, but neither yellowish nor greenish.

Chemical Tests.—The most practical test for fat is Lewi's modification of the Babcock test, which requires special but not complicated apparatus, and is performed as follows:

- 1. Place in the flask 2.92 c.c. of milk as measured in the special pipette.
 - 2. Rinse the pipette thoroughly.
- 3. Add gradually an equal quantity of chemically pure sulfuric acid, mixing with the milk by gently rotating the flask; the color changes to dark brown through oxidation of the sugar.
- 4. Add 0.6 c.c. of equal parts of amyl alcohol and strong hydrochloric acid.
- 5. Add 50 per cent. sulfuric acid in sufficient quantity to bring the fluid up to the neck of the flask.
 - 6. Centrifuge three or four minutes.
- 7. Read off on scale; each 0.1 on the scale represents 0.3 per cent. of fat.

In giving this test in full, we must not fail to note what is generally omitted, namely, that extreme caution should be exercised in adding the pure sulfuric acid, and afterward in diluting the same acid with water to a 50 per cent. solution; the acid should always be added gradually to the other fluid, otherwise great heat is produced, resulting in possible accidents, or at least impairment of the value of the test.

The best test for the proteins, Kjeldahl's, is not at the command of the ordinary practitioner. Koplik¹

recommends a modified Esbach test as proposed by Woodward. Two milk burettes, each containing 5 c.c. of milk, are allowed to stand over night at a temperature of 100° F. They are then cooled. The milk is drawn off into two Esbach tubes and 10 c.c. of the Esbach reagent are added. The tubes are then shaken, and centrifuged until the reading is constant. This test registers the total proteins.

The percentage of milk-sugar is so constant as practically never to require quantitative testing.

RULES FOR BREAST-FEEDING.

The nursing mother should lead a quiet and regular life, avoiding social diversions that involve late hours, especially if housework is demanding a great deal of her attention. We have seen that the nature of her diet affects her milk only slightly, if at all, provided that it is sufficient; in case of great poverty there may be some difficulty on this score, and the relief of nursing mothers is one of the worthiest charities. Regular and moderate exercise is beneficial, helping to avoid one of the great difficulties often attending this period, namely, constipation. This ailment is best relieved by giving such mildly laxative foods as Graham bread, oatmeal, and prunes, but we need not hesitate to give a harmless drug like cascara sagrada. in sufficient dosage, should medication be unavoidable. Alcohol, except in very small quantities, is objectionable, but a glass or two of beer per day will hardly be injurious, and is recommended by some as a mild galactagogue. Water and milk should be taken quite freely, still not in such quantities as to impair the appetite for substantial food. With regard to tea and coffee, great moderation is advisable; excessive teadrinking is almost as vicious a habit as the excessive consumption of alcohol.

In giving medicines for intercurrent affections we should exercise great care, for many common drugs pass into the milk, and thereby affect the child. Dombrowsky found this to be the case with most aromatics, formaldehyde, phenol, turpentine, and iodoform, to which we may add bromin, iodin, the alkaloids, and to a slight extent mercury, salicylic acid, and alcohol (Voltz).

I have already called attention to the desirability of the four-hour feeding interval, but may, at this point, go a little more into details. The old custom of tormenting the mother, on the first day after delivery, by frequently putting the infant to the breast, is happily, though rather slowly, being done away with; it is useless to try to nurse the baby on the first day, as it cannot suck effectively in any event; we may give it a few spoonfuls of water, as previously suggested. Twenty-four hours after birth we may begin with the regular four-hour schedule, beginning at 6 A.M. and ending at 10 P.M. Night-feeding is usually unnecessary, is largely based on tradition, a great hardship to the mother, and of doubtful benefit to the baby; it is pleasing to record that it is gradually being abandoned. Thus the infant obtains five meals per day, which experience has shown to be usually sufficient. There are some cases, however, in which the infant seems to draw a somewhat inadequate supply, and the stomach is empty before the expiration of two hours and a half. Rosenstern¹ thinks that, in this event, we may safely make a slight concession and nurse the baby at three-hour intervals, six or seven times a day; another plan would be to give a night feeding at 2 A.M., during the first few months, but Rosenstern's method seems to me the better of the two.

The duration of each feeding is usually set at twenty minutes, but this rule is arbitrary and not justified by the physiological facts, for Feer has shown that most of the meal is ingested in the first few minutes. A better plan is to observe the baby during nursing and to take it from the breast as soon as it ceases to suck actively; this method has the additional advantage of giving us a fair index of the milk-supply. If the baby draws only a short time, the nipple being in normal condition, there is reason to suspect a scanty flow of milk; on the other hand a vigorous infant may easily empty a full breast in ten minutes and then stop sucking, being perfectly satisfied; in this case there can be no object in keeping it any longer at the breast, to fall asleep there.

I have referred to the prophylaxis of fissured nipples, consisting in keeping them clean and dry; the best method is to wash them daily with a saturated solution of boric acid and dry them gently with sterile gauze. This procedure should be begun some months before delivery, and at the same time the development of

badly formed nipples may be aided by daily gentle traction and manipulation, best performed by the prospective mother herself. After delivery, similar cleansing should take place after each nursing, and the nipples be protected from the clothing by small pads of sterile gauze. Should a fissure appear in spite of these precautions it should be treated with silver nitrate and the child temporarily restricted to the unaffected breast; in this way the development of a mammary abscess may usually be avoided. unused breast is apt to cake from the stagnation of milk; for the relief of this condition we must resort to pumping and gentle massage to keep the breast from going dry, the caking under control, and to guard against inflammation of the affected gland. difficulties just referred to are especially likely to occur at the very commencement of lactation, and in primiparæ.

In regard to the care of the infant's mouth a revolution in practice has taken place in recent years; the rule now reads: let the baby's mouth alone. We have repeatedly observed the erosions on the palate, called Bednar's aphthæ, in infants whose mouths were regularly and conscientionsly rubbed after each meal with solutions of borax or boric acid. The healthy breast-fed baby has a mouth that keeps itself quite clean enough; the surest way to infect it is to rub off the protecting epithelium by unnecessary attempts at cleansing. Pacifiers and teething-rings are simply abominations; they cannot be kept clean, and are used by the infant to convey dirt of every kind into

its mouth. We might, in this connection, call attention to the belief of Pedley, that the continued use of the rubber pacifier leads to deformity of the jaws; while this is open to doubt, the other objection, referring to the extreme dirtiness of this popular infant sedative, cannot very well be challenged. In passing we may mention, as other means of infecting a healthy mouth, the performance of unnecessary operations on the frenum linguæ and the edge of the gums, both still too frequently practised by physicians of repute. Let us remember that true tongue-tie, sufficiently severe to interfere with nursing, is quite a rare condition, and that the eruption of the teeth is a physiological process, which may give rise to some slight discomfort and irritability, but is never the cause of actual illness. Among the hundreds of infants brought to me with the home-made diagnosis of teething, I have never failed to find some disturbance of the digestive or respiratory tract to account for the symptoms present.

DIFFICULTIES IN BREAST-FEEDING.

The principal and most frequent error committed in the nursing of infants is overfeeding, usually accomplished by adhering to the old tradition of nursing at very frequent intervals, of two hours or even less. Sometimes the anxious mother nurses the baby whenever it cries, thinking that it cries from hunger; as overfeeding leads to intestinal colic, which causes the infant to scream loudly, it is evident that a vicious

circle is easily established. Nevertheless, overfed nurslings usually thrive tolerably well, in marked contrast to overfed bottle-babies, but they do not gain in weight in proportion to their intake, and regularly suffer from moderate diarrhea, with three or more rather thin and often green stools daily. If a robust, breast-fed child has more than two or three intestinal movements daily we may safely make a tentative diagnosis of overfeeding and investigate the case accordingly. These babies constitute the majority of partial failures in breast-feeding; in my dispensary service at least one-half of all the breast-fed infants present the above picture, though we must remember that babies who are really thriving are not brought to the dispensary at all, so that the true ratio is probably much less. These infants improve rapidly, and their diarrhea and colic cease within a week or two if the feeding interval is lengthened to four hours, and the invariably present night-nursing cut out. It is often useful, though rarely necessary, to precede this change of régime with a dose of castor oil, and a twentyfour-hour period of starvation, in which only water may be given, so as to give the overloaded and overworked intestine a cleansing and a brief period of rest.

More serious is underfeeding. In mild cases, due to a moderate insufficiency of the milk as a whole or the milk fats, the prominent symptoms are constipation and a rather slow gain in weight. These infants are best kept at the breast, for the first months at least; they are not likely to do much better on artificial feeding, and may not do as well. A few teaspoonfuls of oatmeal water, given before each nursing, may aid to keep the bowels moving; if this is insufficient we may give an occasional small dose of castor oil. The administration of calomel or, still worse, rhubarb is altogether out of place in this condition. In some of these cases the three-hour interval, before noted, may be tried. I doubt if the results are worth mentioning, and personally have seen no benefit therefrom.

When the mother's milk supply is seriously deficient there is little or no gain in weight, and sometimes even a loss; the infant cries almost continually and passes small, dark green stools, with some mucus; these are the so-called hunger-stools, consisting largely of intestinal detritus and bacteria, with a relatively small proportion of the end-products of milk digestion. Under these circumstances we must usually resort to artificial feeding; but in the first month of infancy, if the starvation is not severe and there is ever so slight a gain in weight, we do well not to be discouraged too soon. Breasts that secrete poorly at first may eventually furnish a moderately good supply of milk, for at any rate a few months, thus tiding the infant over the most critical period. Wile observes that sometimes the breast may be induced to afford sufficient milk, after several weeks of forced suspension of function: similarly a gland that begins poorly, especially in a very young mother, may become, with patient stimulation by means of regular application of the infant, a very fair purveyor of nourishment.

If a baby seems to obtain sufficient food and yet manifests a deranged condition of its digestive appa-

ratus, as if its diet disagreed with it, the trouble may be with the quality of the milk, but is more likely to lie with the child itself. This is, indeed, the clinical picture of what Czerny² first described as the exudative diathesis, which we shall discuss at length in a later chapter. At this point we merely wish to emphasize what was urged by Czerny² himself, namely, that slowly as these infants get along on breast-milk they invariably do worse on artificial food; in short, the only safe procedure is to keep the child at the breast at all hazards, regardless of the fact that it is not doing as well as might be desired. As to inferior quality of the breast-milk, we have seen that this factor is not easily determinable and often doubtful. may, in some cases, manage to make out a serious defect through our physical and chemical examination; at other times, however, we may be dealing with some obscure constitutional disturbance of the mother, manifesting itself in the milk, but defying determination by laboratory methods.

The social difficulties in the way of breast-feeding play a great role, but may eventually be obviated, or at least reduced from their present wide scope, if the public at large will give the medical profession a helping hand. In recent years it is pleasing to note that society women, after decades of almost total neglect of the first duty of motherhood, are beginning to realize the proper state of things, so that breast-feeding is gaining ground rapidly in the higher walks of life. The longer feeding interval, combined with the eighthour rest at night, is doing much, and will do more, to make maternal nursing popular and even fashionable. Among the working classes the situation is more difficult, and can be saved only by the general acceptance of the long feeding interval; it must also be fostered by appropriate legislation on the subject of female labor, in which France has led the way, and most of the United States lag far in the rear. Much was expected of cash nursing-premiums in France and Germany; their value has been questioned in the countries of their adoption, and Herrman earnestly warns us against experimenting with this social placebo. Far better is a medically controlled system of education by visiting nurses, now being tried out on a fairly extensive scale in some American cities. maternity hospitals are doing excellent work in following up discharged infants and encouraging their mothers to keep them at the breast. These measures cannot. however, be more than palliative, unless suitable labor legislation actually puts a premium on nursing by making it economical; if this is done there is not the least doubt that the majority of mothers will prefer it and artificial feeding will drop from its present prominence to a secondary position.

WET-NURSING.

When the mother is physically debarred from nursing her infant, a good wet-nurse should afford a fairly adequate substitute; unfortunately, in this country, a good wet-nurse is a *rara avis*, in fact, nearly unobtainable outside of our largest cities. We have no sturdy peasantry to draw upon, as is the case in many parts of Europe. Our prospective wet-nurses are chiefly drawn from the very dregs of the city slums, are unfit to dwell under the same roof with decent people, and relatively often diseased. These circumstances have led most American practitioners to advise against the employment of wet-nurses, except when absolutely unavoidable.

In selecting a wet-nurse, one desideratum is that her delivery shall not have preceded the birth of the prospective nursling more than a few weeks, or a month or two at the most; the milk secreted during advanced lactation is likely to be too rich for the newborn infant and lead to indigestion. It is, furthermore, important that she should yield an ample quantity of good milk; this may be readily determined by a careful examination of herself, and inspection of her own infant, the condition of the latter being a good index of the prospective milk-supply. It is indeed inadvisable to engage a wet-nurse who cannot show a robust baby of her own. The third requirement is a searching investigation of the nurse's health, with special reference to the absence of syphilis and tuberculosis. As to syphilis, in addition to the examination for luctic stigmata of mother and child, we have in the Wassermann test as good a criterion as could be desired if it is made by an expert and interpreted in connection with the physical findings; the loss of time involved in the performance of this test is amply compensated by its trustworthiness. As to tuberculosis we are still chiefly dependent on physical examination, which is not

absolutely conclusive, and we must be largely guided by the woman's general condition. The v. Pirquet test is a little too delicate for our purpose, as it reacts positively to long since healed tuberculosis, as well as to the non-infectious glandular forms of the disease. Many possibly eligible wet-nurses cannot meet this exceeding searching test, which would make the final selection of a suitable person difficult if not impossible. At the same time a negative v. Pirquet reaction is a good guarantee of the absence of even latent tuberculosis, and therefore unquestionably of the greatest value.

PRESERVED BREAST-MILK.

Certain foreign authorities, in close touch with large maternity hospitals, have suggested the preservation of surplus breast-milk for the benefit of babies whose mothers are unable to nurse. To preserve milk, Budde takes advantage of the catalytic ferment present in fresh human milk, to split hydrogen peroxid and sterilize the milk with the nascent oxygen. Budde's method is carried out as follows. After neutralizing a liter (quart) of milk with sodium bicarbonate, which may even be in slight excess, he adds 0.5 gram (8) minims) of a 30 per cent. (100 volume) solution of hydrogen peroxid, the best preparation being the one sold under the name of Perhydrol. The mixture is then warmed to 55° C. (131° F.), so as to liberate nascent oxygen freely. Milk thus treated will keep perfectly fresh for twenty-four hours, when the procedure must be repeated. Peiser has thus managed to

preserve milk for as long as fifty-two days. Mayer-hofer and Pribram recommend this method highly, and it certainly seems well worth trying, whenever a non-nursing mother can get into communication with another woman who has a surplus of milk at disposal. One conspicuous advantage is that it obviates the necessity of the wet-nurse abandoning the breast-feeding of her own child; another is, that the donor can remain in her own home; the third lies in the availability of a higher grade of wet-nurse; the fourth is the circumstance, that the milk can be transported a considerable distance.

The milk must, of course, be drawn with a pump and fed from a bottle; this means that all the rules of cleanliness must be enforced that are required with the handling and feeding of cow's milk. Under these conditions the plan here outlined will often be invaluable, and there are many cases in which it can be tried before resorting to the last refuge of feeding with cow's milk. Hoobler³ is the most recent advocate of the collection of surplus human milk for use in hospitals and dispensaries, and the peroxid method should be a valuable aid in the successful accomplishment of this purpose.

CHAPTER III.

COW'S MILK.

Practically the only available substitute for human milk is cow's milk. In some countries the milk of other animals has been given to infants, but in America the cow is the one prevailing source of supply.

Cow's milk is a faintly acid fluid, of a yellowishwhite color, that separates, on standing, into two layers. Within four to eight hours these layers are sharply defined, the upper very opaque, of a decided "creamy" yellow tint, containing nearly all the fat, and the lower translucent, bluish white, and nearly fat-free. The depth of the upper layer is more or less proportionate to the percentage of fat in the milk.

Milk is sometimes turned blue by the Bacillus cyanogenes (Hammer); this alteration appears to be harmless.

Composition.—The composition of milk obtained from Holstein or "grade" cows ranges about as follows:

Water		87.0	to	88.0	per	cent
Proteins		3.0	to	3.5	-	
Fats		3.5	to	4.0		
Milk-sugar				4.5		6.6
Salts				0.73	5	

The total caloric value of ordinary cow's milk ranges from 620 to 680 per liter (quart), or 19 to 21 per ounce, practically the same as of human milk. Clinically

the inferior digestibility of cow's milk calls for a reduction from the above estimate of about 30 calories per liter, or 1 per ounce.

Cows of the Guernsey or Jersey breeds give a far richer milk, with 4 per cent. of proteins, 5.5 or more of fats, and 5 of sugar, a caloric value of 850 or more to the quart, 25 or more to the ounce.

It will be seen that the proteins are far more abundant than in human milk, the carbohydrates about as much less, and the fats about the same. The salts are nearly four times as abundant, and are divided about as follows (Pelka):

Phosphoric acid	0.21 per cent.
Lime	0.15
Potash	0.18 "
Soda	0.10 "
Chlorin .	0.11 "
Iron (as oxid)	0.3 to 0.7 mg. per liter

It will be seen that iron forms an exception and is considerably scantier than in human milk (Langstein¹).

Van Slyke calls attention to the fact that the salt estimates do not allow for the salts of the organic acids, which would raise the total salt percentage to 0.9; this happens because the salts are always calculated as ash, and the organic acids become converted into carbon dioxid, which escapes during incineration.

Of minor but still not negligible importance are the ferments, and the greatest possible interest attaches to the bacteria found in cow's milk.

Analysis of Cow's Milk.—The fats may be estimated by the method given for human milk. Bowditch and Bosworth obtain a complete analysis in the following

simple manner, and have found the results, thus calculated, sufficiently accurate for all practical purposes:

Let L= the reading of the Quevenne lactometer (specific gravity -1000) and let F= the percentage of fat: Then

```
\begin{array}{l} \frac{L}{4} + (1.2 \times F) = \text{total solids.} \\ \frac{L}{4} + (0.2 \times F) = \text{solids not fat.} \\ (F - 3) \times 4 + 2.1 = \text{casein.} \\ (F - 3) \times 4 + 2.8 = \text{total proteins.} \end{array}
```

Total solids—(F + total proteins + 0.9) = sugar, (0.9 is added) to allow for the salts).

The Proteins.—Most authorities calculate the casein as about three-fourths of the total proteins and lactal-bumin and lactoglobulin, the so-called whey-proteins as one-fourth. Grulee¹ thinks that the latter have been overestimated and rates them as only one-sixth or even less. This difference he attributes to inaccuracy in analytic methods, inasmuch as most investigators do not coagulate the casein perfectly, so that some of this protein drains off into the whey, and is erroneously estimated as lactalbumin. A few years ago this diversity of opinion would have been regarded as of some moment; in the present state of our knowledge as to the assimilation of the proteins, the matter may be considered of minor and perhaps only academic importance.

Until quite recently the casein of cow's milk was almost universally regarded as less digestible than human casein, and this belief was based on the following observations: (1) cow's milk contains about twice as much casein as human milk; a priori, it would seem

that the former would be apt to overburden the infant with this protein, even if somewhat diluted with water: (2) it was noticed that cow-milk casein coagulated in the baby's stomach in large, hard masses, whereas human casein formed delicate flocculi: this was attributed to a chemical difference between the two forms of casein, which appeared well established by the investigations of Siegfried and others, but, as a matter of fact, is not absolutely proven. Alexander and Bullowa give a purely physical explanation for the difference in coagulation; they call attention to the fact that case in is an unstable colloid, protected by lactalbumin, and that this protection is more effective in human milk, which contains a far larger proportion of the latter proteid; for the same reason the human casein naturally occurs in a much finer state of subdivision, and therefore forms a more delicate coagulum, when attacked by the rennin of the infant's stomach. There is no reason to doubt the accuracy of these observations, and we are forced to admit that the coarse coagula of cow's-milk casein are more difficult of digestion. This disadvantage may, however, be avoided if we follow the advice of these authors, which merely repeats the recommendations of a past generation. If we protect the cow'smilk casein artificially, by the addition of a mucilaginous fluid like barley-water, the coagulation of this substance is rendered very similar to that of human casein, and the formation of large unmanageable masses is effectively checked. We may add that Langstein² has shown that human and bovine casein

are peptonized exactly alike by the infant's digestive secretions.

In this connection we may well devote a paragraph to the subject of casein curds in the stools. Some years ago all more or less white lumps and particles in the feces were called curds by American practitioners, though the term curds is properly applied only to coagula of casein or, at most, proteins. Meanwhile many foreign observers claimed that casein coagula never occurred in the feces, and that the white particles referred to were composed almost wholly of fats and fat derivatives. A brisk discussion ensued: Talbot² ³ represented the faction, chiefly American, who insisted on the frequent presence of true casein in the feces, whereas the opposition, chiefly German, who denied the occurrence of true curds in toto, were represented in American pediatric literature by an article by Meyer and Leopold aside from many transatlantic contributions. Southworth and Schloss tried to take up an intermediate position by admitting that some fat was mechanically included in the casein curds, a useless concession of a recognized fact, which did not touch the main issue. Finally, Brennemann¹ showed conclusively that the two parties had been discussing two entirely different things; that the small, soft, yellowish particles, referred to by the German school, but quite as well known here, were not really curds at all but actually fatty bodies; on the other hand, the large, hard, grayish-white concretions, discussed by Talbot, well known here, but almost unknown in Germany, were true casein curds, containing a large proportion of fats, as curds derived from whole milk invariably do. Talbot's contention is now admitted to be correct by a number of German observers, for example, Bauer² and Ibrahim,² who acknowledge that the hard curds consist chiefly of undigested casein. As to the cause of their appearance in the stools, Brennemann¹ claims that they follow the ingestion of unmodified or merely diluted raw milk, thus bringing the subject in close relation to the discussion in the preceding paragraph. They are rather rarely observed in Germany, because the Germans seldom feed babies on raw milk. As to their real importance. Brennemann rates it low, but thinks that they may cause some slight intestinal disturbance; they undoubtedly represent a loss to the infant of a certain proportion of the food proteins, and are objectionable on that account, if no other; at any rate they are usually avoidable by simple measures, the adoption of which I think advisable, and which shall be taken up under the head of milk modification.

The whey proteins, lactalbumin and lactoglobulin, are also digested about as well from cow's milk as from human milk. A few years ago this fact was seriously doubted, but this doubt rested on a misapprehension regarding certain experiments with whey-feeding. Meyer² disposed of this question by proving that any trouble with whey-feeding could not be attributed to the proteins, and recent investigators, such as Bickel and Roeder, are satisfied that the whey proteins of cow's milk are a perfectly digestible infant food.

Summing up, we see that the absorption of the cowmilk proteins is little, if at all, inferior to that of the proteins of human milk; in other words, it is nearly perfect. The high percentage noted on page 23 was obtained with artificial feeding, and it is hard to see any room for improvement.

The Fats.—The chief difference between the fats of human and of cow's milk is the excess of oleates in the former, and the high ratio of volatile fatty acids in the latter; the volatile fatty acids, especially butyric acid, are six to eight times as abundant as in human milk (Stern). These acids, if they exceed a small amount, are, as we have seen, irritating to the infant's intestine; they are, moreover, readily converted into bodies of the acetone group, the toxic action of which will be referred to later. In observing that the fats cause the most serious difficulties in the substitution of cow's milk for human milk, we must not forget that the newborn calf is about as far advanced in development as an infant aged nine to twelve months. Now babies of that age rarely have any trouble in digesting cow's milk; it is, in fact, their usual and staple food, and they thrive on it exceedingly well. In the case of young infants, on the other hand, it is evident that cow's milk is a rather unsuitable food, and the greatest difficulty is probably afforded by the volatile fatty acids. Even though these acids amount to only 3.5 to 4 grams per liter, this represents an excess of fully 3 grams over the amount in human milk, and cannot be an indifferent matter in view of what has been said. The differences between the two kinds of milk, as regards the other fatty bodies, are so much less in degree and kind that they probably play a minor part.

Bahrdt has shown that even in infants who take kindly to cow's milk the ratio absorbed is only about 83 per cent.; this is at least 10 per cent. less than the proportion absorbed from human milk, and represents a deficit of about one calorie per ounce. This unabsorbed fat combines with the milk calcium to form the calcium soaps, which make up so large a part of the feces of artificially fed infants. It is true that cow's milk contains a considerable surplus of calcium which is not needed; it is, however, not alone this surplus that is bound by the free fatty acids, but part of the calcium required for the body's growth is also lost in this way. The deficit in fat has therefore added to it the still more serious deficit in lime salts. which does not appear in the caloric calculations, but is of far greater influence on the infant's ultimate welfare.

The disadvantages, so far recorded, are still within what may be called physiological limits, though certainly not perfectly normal; the succeeding chapters will show that these limits are easily passed, and that the line between what is physiological and what is pathological is not easily drawn in artificial feeding.

The Carbohydrates.—The lactose of cow's milk is chemically identical with that of human milk; its digestion and assimilation should not, therefore, present any differences. As a matter of fact the assimilation of sugar is often disturbed in artificial feeding,

but this is invariably secondary to some other digestive derangement so far as milk-sugar is concerned. As to the digestion of other carbohydrates, much used in milk-modification, we need not concern ourselves at this point, in discussing the properties of pure cow's milk.

The Salts.—Sodium and potassium chlorids are respectively five and two and a half times as abundant in cow's milk as in human milk. They are absorbed in somewhat smaller ratio, but in acutally larger amounts, so that there is a tendency to overload the organism with both potassium and sodium salts; there is, therefore, a markedly greater excretion of both, especially through the urine, in bottle-fed than in breast-fed babies. Under normal conditions this is of comparatively little consequence, but under pathological conditions the metabolism, especially of sodium, may become a serious matter; we shall have occasion to study clinical pictures in which the absorption and assimilation of these large amounts of salt play a very important role.

We have referred particularly to the part played by the sodium salts, because the potassium salts are supplied by cow's milk in a more moderate ratio and the elimination of any excess is effected more easily and rapidly. As a matter of experience, potassiumpoisoning plays a small part in infant pathology, with the doubtful exception of certain metabolic disturbances, such as scurvy, where the role of potassium is not proved and most likely negative, as will appear later.

We have already discussed the main points of calcium

metabolism in connection with the fats; its relation to the metabolism of phosphorus is equally important. The total of phosphorus is five times as great in cow's milk as in human milk, and while its rate of absorption lags somewhat behind, it still remains much greater from the former. It is quite a different matter, when we turn to the retention of phosphorus. Keller has shown that the only assimilable forms of phosphorus, namely, the nucleophosphides, equal 40 per cent. of the total in human milk, and only 6 per cent. in cow's milk, so that the amount of organic and available phosphorus per liter of human milk is actually greater, 0.16 gram against 0.12. We have seen that inorganic phosphorus, as in calcium phosphate, is not capable of assimilation. Thus the retention of this element is inevitably somewhat lower in cow-milk feeding, and the great bulk of unavailable phosphorus passes off in the feces and urine. Furthermore, there is not sufficient phosphorus absorbed to combine with the supply of calcium in the tissues. Although, as we have seen, there is an enormous waste of calcium, still the amount absorbed is in excess of what is required to bind the available phosphorus. The result is, that an additional quantity of calcium is excreted in the lower intestine, to increase still further the already large content of lime-soaps in the feces.

We have noted the scanty supply of iron in cow's milk, quite insufficient to keep up the normal iron metabolism of the infant. This deficiency is made worse by the tendency of the iron to follow the calcium in its extremely wasteful economy. The wonder is

not that artificially fed children are almost invariably anemic, as shown by the hemoglobin test, but that they are not more so.

The Ferments.—The ferments of cow's milk differ considerably from those found in human milk. Diastase seems to be entirely absent from the former, glycolytic ferment abundant, and catalytic ferment scanty (Friedjung and Hecht). These ferments are impaired or destroyed by heating the milk above 55° C. (131° F.), but not by chilling or even freezing. This circumstance has been made much of by the advocates of raw milk feeding; we have, however, seen in the preceding chapter, that the milk ferments play, at the most, a very subordinate part in the nutrition of breast-fed infants, and there is no evidence, that they are of any importance in feeding with cow's milk. It is being more and more generally admitted that the insistence on feeding with unheated milk, not long ago almost universal in this country, and largely based on the above premises, rests on a very unsubstantial foundation. It is at present exceedingly doubtful if the ferments of cow's milk have any influence on the food value of that fluid.

MILK BACTERIA.

Bactericidal Properties.—Probably because of the contained ferments, fresh milk does exercise some restraint on the growth of contained bacteria. Coplans found that this quality persisted, in a diminishing degree, for twenty-four hours, if the milk was simply left standing

at the room temperature. Chilling to the freezingpoint impaired this faculty, so that bacteria began to grow freely after nine hours; exposure to a temperature of 38° C. (100° F.) started free growth in six hours. Evidently there were two factors at work, on the one hand, the inhibiting power of the ferments, on the other, the thermic conditions of bacterial development. Hunziker found that the bactericidal or, better, inhibitory power, was most pronounced at 21° C. (70° F.). We shall recognize the full importance of these observations presently when we come to the question of the milk-supply. Otherwise this matter must be admitted as of minor importance, especially as the inhibitory action of fresh milk on bacteria is limited both in degree and duration.

Milk Bacteria.—Whereas breast milk is ingested in an almost germ-free condition, this is impossible with regard to cow's milk under the most favorable circumstances. Milk forms one of the best imaginable culture media for countless harmless as well as pathogenic microörganisms, as is evident from its composition, comprising, as it does, all the food elements. When we have in addition a temperature near bloodheat, as happens very often in the summer months, conditions are perfect for the growth of many of the most virulent germs. The elaborate investigations of Park and Holt differentiated no less than 239 species of saprophytic bacteria growing in milk; they found that ordinary store milk, sold from the can, might contain more than 20,000,000 bacteria to the cubic

centimeter; bottled milk, though far cleaner, not rarely contained 500,000 germs; whereas by observing reasonable precautions, milk containing less than 10,000 was easily procurable. They found that it was quite feasible to furnish a nearly germ-free milk by adopting proper care in milking, and adequate provisions for keeping and transportation, and decided that milk containing above 1,000,000 bacteria to the cubic centimeter was unfit for human consumption.

Milk Infection. More serious than saprophytic bacteria, which merely decompose the milk and cause illness through the production of poisons and toxins, are the recognized germs of infectious diseases, which may enter the milk through the most varied sources of contamination and find it a particularly effective culture medium. The literature on milk-borne epidemics of disease has recently grown to large proportions; we may here note the salient facts.

For momentous and striking consequences, milk-borne typhoid fever heads the list. This source of infection caused hundreds of fatalities, in the city of New York alone in 1912 and 1913, though ordinary precautions had been taken and the infected supply was speedily cut off. In many of our cities, contaminated milk has become the main source of this disease, which the most stringent regulations cannot eliminate entirely, so long as raw milk is consumed. After weeding out all other sources of infection, there still remains the danger of contamination by typhoid carriers, which cannot be guarded against with absolute certainty, under the best of supervision. The only

preventive, that is infallible, is sterilization or efficient pasteurization of the entire milk-supply, as closely as possible to the time when it is handed to the consumer.

Second comes tuberculosis, the germs of which are easily conveyed from the tuberculous cow to the milk, even if no bacilli are directly excreted by the udder. In addition there is the less frequent, though by no means negligible risk, of infection with human tuberculosis from one of the many persons who handle the milk. Even the latter source is not easily controlled; the former, that is ever present, and can be eliminated only through destruction of diseased cattle, is at present taxing our state authorities to the utmost, with hitherto very unsatisfactory results. The tuberculin test shows that a very large percentage of our cows suffers from tuberculosis, and that the ratio tends to increase, in spite of the elimination of infected animals; carried out thoroughly, it is almost ruinous to the dairying industry, and a heavy burden on the State, which must pay the farmer for the cows that are condemned. The question has often been raised, whether the danger is sufficient to warrant such drastic and costly measures, especially since the bovine and human strains of tubercle bacilli have been differentiated. Theobald Smith claims that the danger is trivial unless the cow's udder is infected; nearly all authorities agree that the bovine bacilli cannot cause the familiar type of pulmonary tuberculosis. This last proposition, however, by no means disposes of the matter, for the forms of tuberculosis, here in question, while not as

severe as those caused by the human bacillus, are nevertheless serious, especially in children, and sometimes have a fatal issue. Kober reports no less than 86 cases in which tuberculosis could be traced to milk; Raw claims great frequency for infection with the bovine type, and Bovaird shows that these forms of tuberculosis are much commoner in England than American experience would lead us to suspect. Park,² in a careful investigation, shows that about 2 per cent. of the fatal cases of tuberculosis in New York City are caused by the bovine bacillus; this looks like a small ratio, but signifies about two hundred deaths per year, a number that is certainly not negligible.

Thus the possibility of tuberculous infection by bovine bacilli, of course through the alimentary tract, must be accepted as a fact, which would be even more conspicuous if the mass of the population were not in the habit of boiling all milk. Behring's stand-point, that infection with tuberculosis usually takes place in infancy, and through the digestive tract, may be, in some respects, too sweeping; so far as infection by bovine bacilli is concerned, however, there is today little doubt as to the correctness of his views.

Many acute infectious diseases of other kinds are conveyed by milk. Thus, Hemenway reports an unquestionable epidemic of milk-borne scarlet fever; Brush describes the spread of foot-and-mouth disease, from infected cattle through the milk. Similar reports have been furnished by other observers. Still more interesting is the subject of throat infection with streptococci, derived from infected cows. Reed and

Ward were the first to call attention to the significance of streptococci in the milk, showing that they were derived from mastitis in the cow; then Winslow and others reported a severe epidemic of sore throat in Boston and vicinity, which was easily traced to infected milk. Davis² finally cleared up the bacteriology of this disease by identifying the streptococcus in the human and bovine subjects.

Most important of all, for us, is the infection of milk with bacteria of the dysentery group; this matter is still somewhat obscure in respect to the precise occasion and mode of transmission; the only certainty is that in warm weather a growth of bacteria takes place in impure milk, the ingestion of which causes dysentery in infants. The essential links in the chain are a high temperature and humidity of the atmosphere, to favor the growth of these special bacteria, and an impure milk-supply, swarming with germs ready to assume a luxuriant growth. The best proof that bacteria are essential to this type of infection is afforded by the fact that the morbidity is reduced to an astonishing degree by feeding the infants on milk that is either poor in germs or has been more or less sterilized. As to the symptomatology, pathology, and treatment of infantile dysentery, it forms so important a division of our subject as to merit a chapter by itself later on. At this point it is desirable to give only a few general data.

It is as definitely settled, as anything can be, that impure milk is the cause of most of the extraordinarily high mortality of early infancy. The recognition of this fact is one of the greatest triumphs of modern preventive medicine; with the improvement of the milk-supply, the deeline in the infant death rate has been simply enormous. For example I give the deaths of infants under one year of age in the Boroughs of Manhattan and Bronx (old New York) for July, August, and September, for 1891-3, 1901-3, and 1911-3, taking three-year averages to eliminate the influence of especially warm or cool summers. At the first date the care of infants of the poorer classes was still in what we now would call a deplorable state; the next ten years witnessed an extensive campaign in the education of mothers, with some partial efforts in the direction of milk-stations; the following decade was signalized by a marked improvement in the purity of the commercial milk-supply, rigidly enforced by ordinance. The figures are as follows:

Average of	lnfant population.	Deaths.	Death rate per 1000.
1891-93.	48,000	4170	87
1901-03 .	. 60,000	3244	54
1911-13.	82,000	2564	31

The decrease is not only relative but absolute; the estimated annual saving of lives in the third period is about 4500, a veritable army of infants. Ten years ago I¹ considered the second line of figures quite flattering; they were succeeded by a few years of only slight improvement, so that we seemed to have nearly reached the limit. There is no doubt that the fall of the death rate would have practically come to a halt, had not the city authorities actively taken up the

question of pure milk. The number of deaths for the fourth quarter of the year, the healthiest season for infants, for 1911–3, was 1876, 23 per 1000 living; this may be regarded as the normal, though still somewhat reducible death rate per quarter for infants under one year. It will be seen that the city of New York has succeeded in accomplishing the greater portion of this task.

Milk Regulation.—The regulations laid down in January, 1912, by the Board of Health of New York City have been considered a model for other communities, but have nevertheless been improved upon by the same authority. According to these rules the sale of milk is placed under the following restrictions:

Grade A milk, for infants and children, includes: (1) milk certified to by the milk commissions of the county medical societies; (2) milk subjected to similar supervision by the Board of Health and bearing its guarantee; (3) inspected milk, raw, from tuberculintested cows, averaging less than 60,000 bacteria per cubic centimeter and coming from farms meeting a definite standard as to equipment and methods; (4) selected milk, pasteurized, from farms coming up to a slightly less exacting standard, the pasteurization to be conducted according to rules of the Department of Health and the milk to contain not over 50,000 bacteria per cubic centimeter when delivered to the consumer, the bottles to be marked "pasteurized," the maximum of bacteria before pasteurization being 200,000 and the milk being delivered within thirty hours.

Grade B milk, for adults, includes: (1) selected milk, raw, from cows certified to as healthy by a veterinarian, from farms of a high standard as to method and equipment; (2) ordinary milk, pasteurized, delivered within thirty-six hours after pasteurization.

Grade C milk, for cooking and manufacturing purposes only; all ordinary raw milk.

The insufficiency of the above regulations was demonstrated by the typhoid epidemic of September, 1913. The rules were thereupon modified as follows:

No Grade B or C milk may hereafter be sold raw; this limits raw milk to Grade A, Nos. 1, 2, and 3.

Still more recent regulations provide for the observance of precise temperatures and duration of the pasteurizing process, as follows:

Not less	than	155°	F.	for	at	least	6	minutes
**		152°	F.		4.4		12	**
		148°	F.				18	6.6
**		145°	F.		6.6		20	6.6
**		140°	F.		4.6		30	6.6

It is doubtful if the best raw milk is perfectly safe; Rosenow found streptococci and pneumococci in certified milk. Moreover, Cronheim and Müller found the proteins and fats of raw milk less thoroughly digested, and the casein curds, described before, are a characteristic feature of raw milk feeding, so that the superior food value of raw milk is doubtful. The ferment question is, as we have seen, of uncertain or, at any rate, minor importance. The supposed benefits to be derived from feeding with uncooked milk, when balanced against the dangers, which cannot be warded

off entirely, even with the most painstaking care, render its advantages problematical, even on theoretical grounds.

It seems strange that recent writers, such as Rietschel, Zahorsky, and Schereschewsky, still attribute the high infant mortality, in the summer months, chiefly to the heat itself. Even before the recent improvements in the milk-supply I¹ showed that mere heat was only a contributing factor, calling for special precautions in the care of infants, but rarely causing illness by itself. To charge our troubles to the heat alone would be to give the matter up in despair, for our hot and humid summers are a fixed quantity, with which we are absolutely compelled to reckon. Fortunately, experience has shown that the question of summer mortality is chiefly bacteriological, and bacteriological control of the milk-supply is now saving 4500 infants annually in two boroughs of the city of New York alone. Against so momentous a fact the ablest presentation of theory becomes untenable.

To secure a relatively germ-free milk seems a simple matter until we cast our eyes over a list of the things that must be kept scrupulously, almost surgically, clean. These include the pastures, barns, cows, milkmen's clothes and hands, receptacles for the milk, and cans and bottles for distribution to the consumers. Milk keeps well at 70° F. (21° C.) for a few hours, but must be cooled at least to 50° F. (10° C.), to stand prolonged transportation. Milk is brought to New York City from farms often more than three hundred miles distant, and many of them outside the juris-

diction of the State of New York. Even if all precautions are taken a cow may have developed tuberculosis since her last tuberculin test, a milkman may be an unsuspected typhoid carrier, or there may be carelessness somewhere in the frequent handling of the milk en route to the consumer. The last is specially apt to occur if the milk is brought to town in cans and bottled on arrival, a practice still kept up by some dairies of good standing, but none the less objectionable.

STERILIZATION.

Milk may be absolutely sterilized by boiling it for fifteen to twenty minutes, and rendered sterile, as to pathogenic germs, by boiling for three minutes. Whereas prolonged heating undoubtedly injures the milk, especially the nucleins and lecithin (Baginsky³), the idea that brief boiling impairs milk, as an article of food, is absolutely unsupported by evidence, and may be dismissed: we have seen that the milk ferments. which are undoubtedly impaired, are probably quite unnecessary to the digestibility of the milk. Brennemann¹ voices the general opinion when he claims that, at the worst, boiled milk may sometimes cause constipation; it is highly improbable that, more than raw cow's milk, it is a factor in causing rickets, the danger of scurvy, so widely apprehended, is not involved in ordinary, imperfect sterilization, and can, furthermore, be guarded against by simple methods, to be detailed later. Such eareful observers as Ladd³ and Finkelstein² are convinced that sterilized milk is quite

as digestible as raw milk. Any good thing may, however, be overdone, and I have seen a number of babies, often, be it noted, in the families of physicians, in whom strictly surgical sterilization of the milk had produced scurvy. This sort of sterilization, still more superheating under pressure, seems to impair the milk seriously, and has unquestionably led to the development of the disease mentioned. I may here note that, in former times, when the milk was merely brought to the boiling-point for a very short time, scurvy in infants was rarer than when thorough sterilization was adopted.

Beattie and Lewis suggest the sterilization of milk by means of high frequency currents.

PASTEURIZATION.

Most of the real and supposed impairment of milk involved in sterilization may be avoided by the process known as pasteurization, which consists in heating the milk repeatedly, or for a prolonged time, to a temperature considerably short of the boiling-point. Relay, or fractional pasteurization, gradually kills all the bacteria by destroying them, as they germinate from the spores that have survived the moderately high temperature, at which pasteurization is carried out. Prolonged heating kills all the pathogenic bacteria, but some saprophytic germs survive in small numbers, so that the milk will not keep indefinitely, as will the completely sterilized article. The latter method of pasteurization has gradually supplanted the former,

which is practically fractional sterilization, since we have learned that absolute sterility of the milk is not necessary, and possibly not desirable. There has been considerable discussion as to the most suitable temperature for pasteurization, and the length of time that it should be kept up. Rotch1 advises against temperatures above 68° C. (164° F.) as tending to injure the milk, but de Jong claims that the tubercle bacillus is not killed by a lower temperature than 72° C. (172° F.). We know that the complementary bodies of the ferments are destroyed at 56° C. (133° F.), so that any idea of preserving the latter and destroying the bacteria at the same time is illusory. Schorer thinks that a temperature of 63° C. (145° F.) kept up for thirty minutes fulfils all necessary requirements. The reader will have noticed that the new regulations of the New York Department of Health go into this matter in great detail. Schorer is right in insisting on strict official supervision of pasteurizing plants that work at moderate temperatures, for, under these conditions, the duration of the process is an essential factor in its effective performance. It is now admitted by all that moderate pasteurization, carried out in this way, does not impair the food value of the milk in any particular; in my own opinion, which I follow in my practice, milk treated in this way, if originally of good quality, is preferable to the highest grade of certified raw milk if we wish to adhere to the motto of "Safety first."

Tonney and Pillinger recommend an ingenious plan for keeping milk during long journeys. A vacuum bottle is first heated and then filled with milk warmed to 65° C. (149° F.); from this temperature the decline in the vacuum bottle will only be very gradual. So long as the contained milk maintains a temperature above 46° C. (115° F.) no growth of germs will take place, but if it falls below that point the milk must be consumed at once or discarded. I need hardly mention that this procedure requires careful thermometry, and very cleanly manipulation, especially when the temperature is being tested; carried out properly it should be invaluable for mothers travelling with infants, as the local milk-supplies, especially at the railway stations, are rarely to be trusted, and usually of extremely bad quality.

One flagrant abuse of pasteurization must not be overlooked, but fortunately we can guard against it, and the newer official regulations have not lost sight of it. It consists in the constant temptation to dairies and milk dealers to pasteurize decomposed and even filthy milk so as to make it pass muster as being poor in living germs. Milk that originally contained more than 40,000,000 bacteria to the cubic centimeter has been thus passed off on the public as a tolerably pure article. It stands to reason that such milk remains unsafe in spite of pasteurization; the bacterial poisons contained by such milk are not destroyed by pasteurization and the dead bacteria remain, so that this article of food is almost as dangerous as if it had been delivered in its original state. To try to rehabilitate decomposed milk by means of pasteurization is a dangerous species of fraud, which

cannot be punished too severely. It can be prevented only by official control of the pasteurizing plants, in which inspection is made of the milk that comes in as well as of that which is sent out.

Owing probably to the destruction of the bactericidal ferments, sterilized or pasteurized milk does not keep as well as raw milk.

CONDENSED MILK.

Commercial condensed milk is of two kinds: the first is simply concentrated, by evaporation in vacuo, to about one-fourth of its original bulk; by adding to it three equal volumes of water we restore the original proportions of the ingredients. Thus prepared it is similar to highly sterilized milk in its food qualities as well as in its tendency to cause scurvy, unless precautions are taken. It has the serious disadvantage of not keeping well once the bottle has been opened, and should not be regarded as more than an emergency food. Its continued use, once quite extensive, is to be severely deprecated.

The second kind of condensed milk, sold in cans, is preserved by adding nearly 40 per cent. of canesugar; diluting with three volumes of water we have a sugar content of over 11 per cent. In discussing the digestive disorders of infancy we shall see how totally unsuitable this mixture is for infant-feeding; the sugar is present in a ratio that is unquestionably dangerous and likely to cause sugar intoxication in a very short time. Twenty years ago, when many babies were fed on diluted canned condensed milk, typical

sugar intoxication was exceedingly common in New York at all seasons, not only in summer; it is more than probable that the increasing use of fresh milk is the chief reason why true cholera infantum has become relatively rare. In my early dispensary days we were fairly swamped with such cases, the mortality among them being simply appalling.

I may add that Jordan and Mott find that condensed milk, despite its mode of preparation, is not always sterile, so that one of its supposed advantages is, to a certain extent, illusory.

DESICCATED MILK.

Desiccation is merely condensation carried out to to the limit, and the objections to condensed milk apply here equally. Desiccated milk may be a convenient article for tropical or arctic expeditions—danger of scurvy (!)—but should have no permanent place in the infant's dietary. It is therefore strange to see it highly recommended by a whole school of French pediatrists, headed by Avignaret, but pleasing to note that Variot¹ and his followers visit it with unqualified condemnation. For my part it seems that any extended discussion of this product would be a waste of valuable space.

FROZEN MILK.

Bischoff asserts that frozen milk is not harmful, and regards freezing as a rather good way of preserving milk; fortunately, few authorities agree with him. Shelmerdine reports an epidemic of winter diarrhea caused by feeding infants with milk that had been frozen and thawed out, and is by no means alone in his experience; he specially notes that freezing affects the coagulation of the casein, and it certainly breaks up the milk globules, and therefore affects the digestion of the fats. The objections to freezing do not apply to cooling to near the freezing-point; on the contrary, mere chilling, performed directly after milking, and kept up until delivery to the consumer is one of the best means of preserving milk, and is now insisted on by the official authorities.

PRESERVED MILK.

Our verdict on preserved milk varies according to the preservative employed. Budde's peroxid process, previously described in connection with human milk, is apparently quite unobjectionable, but not effective beyond twenty-four hours, when it must be repeated; it also suffers from being expensive and somewhat difficult to carry out, and is therefore not in general use. Unscrupulous dairymen and dealers prefer the use of ordinary antiseptics, formaldehyde being the favorite; added in the proportion of 0.01 to 0.1 gram to the liter, it is not absolutely bactericidal. so that the proportion added is usually considerably larger. Formaldehyde disappears from the milk in from one to four days without leaving a trace; its chemical detection is therefore often impossible, but it is a safe rule to suspect abnormally germ-free milk of this method of sophistication (Rivas). The addition of sodium benzoate is also illegal; though Grulee and Buhlig are satisfied that even sick infants can take 0.15 to 0.3 gram (2.5 to 5 grains) of this substance daily without harm, it is very probable that this antiseptic interferes with the action of the digestive ferments. The same is true of boric acid, which is employed less frequently. Sodium fluorid is effective in preserving milk only if added in injurious proportions (Mazzeo); Schwyzer has shown that even 1 milligram per kilo of weight is harmful. Of course it must be borne in mind that the chief objection to the addition of preservatives to the milk is the fact that their use covers up negligence in milking and handling, which are the very faults that it is especially important to avoid.

MILK STATIONS.

Until very recently Grade A milk was limited in supply, and high in price; it was therefore obtainable only by the well-to-do, and sometimes was insufficient even for them. The mass of the population had to depend on ordinary commercial milk, now classified as Grades B and C, at that time not distinguished by official tests, and therefore often of exceedingly poor quality. The lowest grades of milk, dipped from open cans in the dirtiest possible manner, were sold at a price so low as to be almost a guarantee of decomposition or adulteration, yet found ready customers in the poorer quarters of our cities. The first thing to do was to supply the masses with a reasonably good grade of pasteurized milk; this plan, inaugurated in

Paris by Variot, with great success, in the establishment of the gouttes de lait and consultations des nourissons, was introduced into New York by Koplik⁵ in 1889, and on a larger scale by Nathan Straus in 1893. It consisted in the opening of so-called milk stations in the poorer parts of the city, where whole and modified milk were sold to the mothers at cost, and advice on the care of infants was imparted by a competent personnel. During the succeeding eighteen years there was a gradual extension of this kind of enterprise on the part of various private institutions and organizations, the key-note being a charitable motive primarily, and public education only in a secondary way. In 1911 the city authorities began to supplement these activities by establishing stations in districts not hitherto provided for, so that at present the city is very well covered.

It is idle to question the enormous value of the milk stations; the tables of mortality, before and since 1893, tell their own story; every city in which the milk station has been introduced reports a rapid and continuous decline in the infant death rate, to figures that twenty years ago would have been deemed chimerical. With all their good qualities, however, the milk stations, at least as now conducted, harbor a number of serious defects. In the first place, while there is ample provision for babies who tolerate cow's milk, the means for handling difficult feeding cases are utterly inadequate. Secondly, the formulas for modifying milk are rigid and unadaptable, fairly good for the majority of infants, but making no concessions to the more delicate and especially the very young babies.

Thirdly, the instruction given at the stations, whether for home modification of milk, or in general infant hygiene, is almost futile if given to ignorant mothers, who are more or less unacquainted with the English language; the distribution of literature to persons who cannot read is particularly absurd. Fourthly, old-fashioned feeding methods even now prevail; the two-hour feeding interval, for example, is still being inculcated at the New York milk stations. Fifthly, there is too little medical control, too much incorrect medical advice is permitted to be given by trained nurses, who are only too willing to grapple with difficult feeding problems which are entirely beyond their ken. Sixthly, last but not least, comes the pauperizing effect of these institutions. Under the new milk regulations it is possible to buy good pasteurized Grade A milk at a reasonable price from reliable dealers; there is no reason why the city anthorities or charitable institutions should continue to give milk at cost indiscriminately to all persons who apply. It is pleasing to know that the authorities, in the city of New York, are seeking to avoid this evil (Baker), but the temptation to make a numerical record is always great.

Haas objects to the term "milk stations" as tending to discourage breast-feeding, as well as to the milk-selling feature; he thinks that they should be called "baby stations," and that the milk should be bought outside; his arguments are surely worthy of careful consideration, and in close accord with my own experience. In my service at the Mount Sinai Hospital

Dispensary most of the difficulties in feeding sick or delicate babies were removed at one sweep when the hospital board assigned a visiting nurse to the pediatric department of the dispensary, and very wisely detailed her from the social service staff of the hospital, so that the dispensary and the social service division could work together through one visiting agent. Home instruction in the modification of milk and the general care of the infant, carried out under hospital or dispensary supervision, is the true solution of the feeding problem among the poor; at the mentioned hospital the results have been so gratifying as amply to atone for the still postponed installation of a milk station, once strongly urged, but now no longer urgent, since the public milk-supply has become so well guarded by official vigilance. An essential part of this scheme is a visit by the nurse, at least once weekly; the baby is brought to the dispensary fortnightly, to be generally looked over, weighed, and have its feeding regulated for the following two weeks. In the case of sickly infants, and such as need special feeding, the appropriate formulas can be drawn up by the attending physician, and the mother taught by the visiting nurse, to prepare the milk accordingly; in these cases, visits and calls should be more frequent, a variation easily carried out. The question of supplying the food gratis, or at cost, can easily be studied out in the individual case, the very individualization being a guarantee against wholesale pauperizing.

This plan does not interfere with the activities of the Department of Health, in inspecting homes and ferreting out neglected and sick infants, which are then referred to the nearest dispensary. The best results can undoubtedly be achieved by the coöperation of both agencies.

WHOLE MILK FEEDING.

After our prolonged discussion of the digestibility of cow's milk, and the means of obtaining it of good quality, we pass on to observe the results of feeding infants on unmodified or whole milk. This is unquestionably a very good food, after the age of nine months, and forms the staple of the infant's dietary during most of the second year; by that time the baby's digestion usually becomes equal to the absorption of the fats that are so troublesome in early infancy, and the gain in weight is likely to be satisfactory, or even above the average. In early infancy, however, as may be surmised from what has gone before, pure cow's milk is a rather dangerous food. It is true that some infants, especially after the first few months, do fairly well on it, and I could mention a series of authorities who do not hesitate to give it after the age of three months. The general experience, however, is decidedly to the contrary; the successes, just referred to, dwindle into nothingness beside the vast volume of disastrous results to young babies, from whole milk-feeding; in fact it seems strange that any pediatrist should still try systematically to feed infants, under the age of nine months, with undiluted cow's milk. The discussions that have filled this chapter strictly refer

only to the food for older infants, and the raw material for the young baby's diet; the latter will be elaborated in the following chapters, where the modification of cow's milk is considered.

MILK IDIOSYNCRASY.

Some infants simply will not tolerate cow's milk, no matter how carefully modified; they may truly be said to manifest an idiosyncrasy. Many authors have assumed that this intolerance represents an anaphylactic reaction to the proteins of cow's milk; to mention but a few, I cite Schlossmann, Moro, 1 and Kleinschmidt in support of this view. stand-point is as follows: The cow-milk proteins are probably not identical with those of human milk, as is shown by the different relation of the casein to the whey proteins and the phosphorus, and still more emphatically by the successful sensitization of animals, by Bordet and Wassermann, through the injection of the milk of different species. Kleinschmidt succeeded in sensitizing guinea-pigs to cow's milk by way of the intestinal tract. Freund4 found that fat-indigestion played no part in true idiosyncrasy. that the condition itself was exceedingly rare, but that his one case also manifested anaphylaxis to tuberculin, whenever cow's milk was given; he suspected the whey proteins as the cause of the reaction, in this point agreeing with Kleinschmidt.

The majority of pediatrists, however, do not accept

the anaphylactic theory of intolerance to cow's milk. Finkelstein³ is skeptical, Langstein² and Meyer³ are convinced that the difference in the proteins plays no role whatever. Lust² lavs down the following requirements for those who would prove anaphylaxis: (1) they must find a reaction to cow-serum in the blood of the affected child; (2) they must prove the passive transmissibility of anaphylactic antibodies; (3) they must show the development of anti-anaphylaxis after the attack. These requirements have hitherto not been met: we have not even any proof that the casein and lactalbumin of cow's milk differ from those of human milk, though the possibility and even probability of this must be considered. The present situation is one of doubt, but the evidence seems to point to the occurrence, in rare cases, of true anaphylaxis, while, on the other hand, many cases of supposed anaphylaxis must be explained otherwise. It is absolutely certain that many infants encounter difficulties, in digesting cow's milk, that do not require the calling in of anaphylaxis, to explain what can be accounted for on much more simple grounds. Anaphylaxis is strictly a protein reaction, and it is now pretty well settled, that the proteins rarely give trouble, even when the other elements of the milk are badly borne; there is no doubt, that former prejudices against the cow-milk proteins have been potent in influencing observers, and that the anaphylactic theory of milk intolerance is losing ground, in all probability being applicable to only very few cases.

MILK OF OTHER ANIMALS.

The difficulties attending artificial feeding with cow's milk have led to a search for more digestible foods and to attempts with the milk of other animals. Goat's milk is in widespread use in some parts of Europe, and is extensively given to infants; examination and analysis have shown it to be very similar to cow's milk, as is indeed to be expected, on zoölogical grounds. McLean, however, has shown that goat's milk is relatively rich in iron, a consideration that might be of advantage in treating and preventing infantile anemia. As goats are relatively rare animals in this country, and there seems to be little inducement to raise them here, the question of substituting their milk for that of cows can hardly come up in a practical way. The milk of mares and asses has been recommended by European authors; both are relatively poor in fat, containing about one-half the percentage present in cow's milk. As the young of these animals are also born in a comparatively mature state of development, it is to be feared that these species of milk are also unsuited to the digestive apparatus of young babies; at any rate, it has not yet been proved that they have any special value for our purpose. In view of the above facts, the idea of replacing cow's milk with that of other animals has hardly passed beyond the stage of suggestion; it is more than doubtful if any advance in infant feeding can be effected along this line.

CHAPTER IV.

MILK MODIFICATION.

We have seen that most infants, under the age of nine months, are more or less incapable of digesting pure cow's milk. For many generations the appreciation of this fact has led to endeavors at modifying this article of food so that the infant might tolerate it and thrive on it.

Milk may be modified in three ways: (1) by simple dilution; (2) by varying the proportions of its constituents; (3) by adding foreign substances. Many well-known methods combine two or all of these.

SIMPLE DILUTION.

Simple dilution may be handled very briefly. It is clearly illogical to reduce all the ingredients of the infant's food merely because one of them is giving trouble; to do so is simply to substitute starvation for indigestion, undoubtedly a fairly good measure for a few days, but sure to be harmful when long continued. Even undiluted cow's milk, if we allow for the imperfect utilization of the fats, is somewhat deficient in caloric value; by merely adding water we diminish the food value still further;

we may increase the digestibility, but only as any starvation diet rests the gastro-intestinal tract. For continued use, this form of modification must unhesitatingly be rejected.

We may therefore pass on at once to the second type of modification, in which the proportions of the ingredients of the milk are altered.

REMOVAL OF CASEIN.

If we add 5 grams (1 teaspoonful) of rennet to 1 quart (liter) of milk, which must be slightly acid, keep the mixture at a temperature of about 100° F. (38° C.) until it separates into a liquid and a solid portion, and then strain it through cheese-cloth, we have practically all the casein, as paracasein, left on the cloth, the filtrate being the fluid known as whey. It is advisable to break up the curd before straining, so as to obtain, as nearly as possible, a precipitate of paracasein alone; nevertheless, about twothirds of the milk fats become entangled in the curd, so that the whey, besides the whey proteins, contains only about one-third of the fats, but nearly all the milk-sugar and salts. As the filtered whey still contains the report it must be heated to at least 140° F. (60° C.) for thirty minutes to destroy that ferment.

The average composition of whey is as follows:

Proteins						$0.8 \mathrm{\ per}$	cent.
Fats .						1.0	
Milk-sugar						4.5	1.6
Salts						0.7	44
Water						93.0	**

As usually made, whey still contains about 0.2 per cent. of casein, because of imperfect precipitation (Grulee).

Whey is a thin, watery fluid, looking very much like skimmed milk; its caloric value is only about 300 per liter, 9 per ounce, barely one-half that of whole milk; it is therefore adapted only to temporary feeding. While its low fat content is of great value in cases of fat-dyspepsia, its employment nevertheless rests on the old fallacy of the harmfulness of casein, and is unsound for that reason. It is not surprising that the enthusiasm for whey-feeding, without any addition, in digestive disorders, so prevalent a few years ago, is now on the wane. For an especially high recommendation of this fluid, see Southworth,2 who does not by any means stand alone; on the other hand, at practically the same date, we find Westcott crediting whey with the causation of colic, and far from enthusiastic. Since then Leopold and Müller have shown that it rather tends to promote sugar intoxication if a serious digestive disturbance is already present, the very condition for which it is recommended. Ladd's4 suggestion to employ whey as a milk diluent instead of water may occasionally be of use, as it avoids the excessive starvation, and more particularly the very low sugar percentage, obtained by adding water alone. A mixture of half milk and half whey gives the following:

Proteins	2.1	per cent.
Fats.	$^{2.5}$	
Milk-sugar	4.5	1.6
Salts	0.7	1.6
Water	. 90.2	1.4

Calories: 490 per liter, 15 per ounce; an important objection is the high ratio of salts, which is an inherent fault of whey-feeding, and probably responsible, in many cases, for the disturbances attributed to the sugar. This subject will be discussed in full in a later chapter.

REMOVAL OF FAT.

If we allow milk to stand in a cool place for eight hours, and remove the upper, yellowish layer with a dipper, we have, as a residue, the fluid called skim milk, the composition of which is as follows:

Proteins	3.5 per cent.
Fats.	0.5
Milk-sugar	4.5 "
Salts	0.7 ''
Water	90.8 ''

Its caloric value is about 360 per liter, 11 per ounce; its food value is therefore slightly greater than that of whey, the ample supply of proteins more than making up for the reduction in fats, and rendering it, from the modern point of view, decidedly superior as a temporary food, in cases of fat-indigestion. Of course, its continued use is sure to entail a loss of weight. Its sugar and salt percentages, especially the latter, are rather high for employment in infants presenting symptoms of intoxication, but for simple fat-dyspepsia, skim milk is among the best temporary foods available.

Removal of Casein and Fat.—This is accomplished by making whey of skim milk instead of whole milk; such whey differs from the ordinary kind, in containing mere traces of fat, not over 0.2 per cent., with a further reduction of caloric value to about 230 per liter, or 7 per ounce. The administration of this fluid is practically equivalent to giving salted sugar-water, as it involves both fat and protein starvation. The danger of inducing intoxication is, if anything, increased by such a diet, and as a routine feeding, for infants suffering from digestive disorders, it is likely to do more harm than good.

Buttermilk.—Buttermilk may be classed with the fat-free types of milk, but differs in containing free lactic acid. It is prepared as follows: To whole milk is added a pure culture of some form of lactic acid bacillus as a "starter;" this germ gradually decomposes some of the milk-sugar into lactic acid; the free acid, in turn, breaks up the milk globules, forming fatty coagula. When these coagula are fully formed, whereof the dairyman judges by experience, the milk is actively churned, and the coagula are thereby agglomerated into the fatty mass called butter, which is strained off, leaving the acid residue known as buttermilk.

Freshly made buttermilk is a slightly sour, thin, liquid, containing the following ingredients:

Proteins		3.3 per cent.
Fats.		0.5 ''
Milk-sugar		4.0 "
Salts		0.7 "
Lactic acid		0.8 "
Water		90.5

The caloric value is about 340 per liter, 10 per ounce. Buttermilk must be immediately pasteurized to kill the lactic acid bacilli, otherwise there will be formed an excess of acid, with a corresponding loss of milksugar. Delay or neglect in this particular is responsible for the frequently excessive acidity of commercial buttermilk, which, moreover, has a marked tendency to undergo putrid decomposition, so as to become unfit for human consumption.

It will be seen that buttermilk differs from skim milk chiefly in a slight loss of lactose, and the presence of lactic acid. Menschikoff does not hesitate to declare it practically equivalent to skim milk. It is, therefore, a very insufficient food for continued use unless other food substances are added to it. Buttermilk should by no means be confounded with acidified milk, which it resembles only in containing free lactic acid; this confusion pervades the whole literature on the subject, renders many articles on the question quite worthless, and has misled the consuming public to a degree that is almost inconceivable. This confusion is all the more unfortunate in that there is every reason to believe that the free lactic acid is a very unimportant constituent in buttermilk, as suggested by Menschikoff. just quoted. On this point we may once more refer to the investigations of Bahrdt and Bamberg, mentioned on page 22, showing that this small ratio of lactic acid is practically without influence on digestion or intestinal peristalsis.

Buttermilk, as a regular food, is never given alone, but always has carbohydrates added to it; we must allow for this in analyzing the reports on this preparation, which otherwise are incomprehensible. These

mixtures have been used for generations in Holland. but their modern general application dates from the communications of de Jager and Teixeira de Mattos, awakening the interest of the Germans, who at once subjected them to a series of thorough clinical experiments, giving them the name of buttermilk-soup. The different authors vary slightly as to the ingredients: Koeppe² adds 60 grams of cane-sugar and 15 grams of wheat-flour to the liter of buttermilk, stirs thoroughly, and boils three times; Cardamitis adds 70 grams of sugar and 13 grams of rice-flour; among Americans, Holt is more cautious in adding carbohydrates, limiting the addition to 30 grams of barley-meal and 8 grams of cane-sugar. These various additions all add a caloric value ranging from 150 to 300 per liter, or 4.5 to 9 per ounce, increasing its food value to near the normal requirement, but involving the greatest possible risks. Tugendreich, for instance, had unpleasant experiences with what he called buttermilk-fever; Finkelstein⁶ has not the least hesitation in identifying buttermilk-fever with sugar intoxication, and his view becomes justified, when we look at the carbohydrate percentages, 11 to 12 in the mixtures of Koeppe² and Cardamitis, and about the same in other cases; Holt's more rational carbohydrate percentage of about 8 is just so much less likely to lead to intoxication.

Turning to reports that are built on erroneous premises, I merely mention Glaessner's as an instance. He thinks the high fat ratio harmful, some of his preparations containing over 2.5 per cent. Of course,

he had not used buttermilk at all, but must have been imposed upon by a manufacturer; the case only goes to show how uncritical are many of the reports of experiences with this substance. As it happens, the low fat percentage constitutes, in the long run, a standing objection to the continued use of buttermilk-soup, even if its caloric value has been made nearly normal.

Overrated as buttermilk has been, we must not ignore its useful qualities. Rommel sees advantages in the fine division of the casein, the tendency of the lactic acid to prevent decomposition—a very doubtful point, this—and the circumstance that less calcium is lost by being bound to fatty acids. He admits that it is too low in fat for continued feeding, and, as usually given, too high in carbohydrates, and that the salt metabolism is not more advantageous than in feeding with ordinary milk, save for the calcium.

REMOVAL OF WHEY.

Albumin Milk.—Separation of the whey is effected by first warming a liter (quart) of milk to 100° F. (38° C.), adding one-half ounce (15 grams) of essence of pepsin, stirring gently, and keeping the mixture at the above temperature for half an hour. This gives us the familiar junket, or curds and whey. We then strain off the whey through linen for half an hour, without squeezing the curd; the whey is rejected. Next we rub the curd through a fine sieve with a wooden spoon, using a pint of water in doing so; this gives us

an emulsion of the curd of a quart of milk, in a pint of water. To this is added a pint of buttermilk; the result is a quart of what Finkelstein and Meyer call Eiweissmilch, i. e., albumin-milk.

We can understand the composition of albuminmilk best, by noting the source of its constituents. It is presumed that the original milk contains 3.5 per cent. of proteins, and 4.0 per cent. of fats.

			From quart of whole milk, grams.	From pint of buttermilk, grams.	In quart of albumin- milk, grams.
Casein			24.0	12.0	36
Whey proteins				4.0	4
Fats			26.0	$^{2.0}$	28
Milk-sugar				20.0	20
Salts .			0.5	3.5	4
Lactic acid				4.0	4
Water					904

If we start with milk containing only 3 per cent. of proteins and 3.5 of fat, the casein and fat will be each about 0.5 per cent. lower in the resulting albuminmilk, agreeing closely with the published analyses of Pelka and others. The caloric value of albuminmilk is about 490 per liter, 15 per ounce; according to Pelka's analysis about 430 per liter, 13 per ounce, neither so very much below a sick infant's requirement, and therefore not involving the disadvantages of semi-starvation, involved in the products before mentioned.

We must remember that the casein curd always includes in its meshes about two-thirds of the fats. Albumin-milk, therefore, is characterized by a very large casein content, rather exceeding that of whole milk, a lowering of the fats by about one-third, and

a reduction of the whey protein, sugar and salts, by about one-half. According to Finkelstein and Meyer,¹ the whey elimination is the essential point, some of the resulting food-deficiency being made up with the additional casein.

Pelka's very complete analysis gives the salts in detail: of a total of 0.44 per cent. he finds 0.13 phosphoric acid, 0.07 chlorin, 0.09 lime, 0.09 potash, 0.06 soda.

The precise technique of preparing albumin-milk is of great importance; the method just described must be carried out with the utmost accuracy. Properly made it is an opaque fluid, filled with fine flakes, which should settle to the bottom very gradually on standing; there should be present no casein lumps nor any separation into layers, except as mentioned. The taste is peculiar, a little but not quite like buttermilk, and some infants do not fancy it at first. Its relatively high caloric value usually keeps the baby's weight from declining; sometimes there is even a slight gain, especially in the earlier period of administration.

Wilcox and Hill recommend albumin-milk made of skim milk. This variation of course reduces the fats nearly to zero, and renders the product very insufficient as a food. It remains, nevertheless, rather superior to ordinary buttermilk, because of its high protein content, and is therefore useful, if we wish to compensate for low ratios of both fats and carbohydrates, by running up the proteins, a plan that is subject to extremely narrow limitations, as we have already seen. Their suggestion may, however, be most profitably utilized, if we subject the milk to partial skimming.

By removing the upper two ounces from milk that has been standing eight hours, we take away about 40 per cent. of the fats; by removing four ounces, we take away about 60 per cent.; in this fashion we can adjust the fat percentage very much as we please to suit the individual case.

Most of the milk modifications, so far discussed, are suitable only for sick babies, being too low in food value for normal infants. In the case of albumin-milk. however, it is possible to continue for some time after convalescence by gradually adding 4 per cent. of carbohydrates, preferably malt; we can thereby increase the caloric value to 600 or 640 per liter, on which a steady gain in weight regularly sets in. The possibilities of albumin-milk, as a regular article of diet, have hitherto hardly been realized; I would therefore like to call attention to papers by Brady² and Benfey, the latter of whom gave it to a large number of newborn infants, with uniformly satisfactory results. I have given it for as long as six weeks, discontinuing it chiefly because the patient no longer required a food that was so troublesome to prepare.

The last feature has been the great obstacle to the general employment of albumin-milk; nurses have to be specially trained to prepare it properly, so that the casein does not gather into lumps that cannot pass through the perforation of the nipple. Engel² tries to obviate this defect by very careful dosage of the coagulating ferments, but practically he only substitutes another technical difficulty for the one mentioned. Hoobler³ has tried the addition of powdered

commercial casein, of the purest obtainable quality, to buttermilk, thus obtaining a fat-free albumin-milk; this plan seems to have some merit, so far as it goes, but the fat-free variety is not usually the kind desired, so that the problem is only partly solved at best.

The details of albumin-milk feeding, especially as they apply to sick infants, are best reserved for later consideration, when we come to discuss the treatment of intestinal diseases.

Albumin-cream-milk.—Quite recently, Feer³ has suggested a substitute for albumin-milk, very similar in composition, with the advantage of being very easily prepared; he calls his product albumin-cream-milk. This mixture is compounded of 500 grams of whole milk, 50 grams of 20 per cent. cream, 10 to 15 grams of Nährzucker (dextrin-maltose), 15 grams of plasmon (sodium-casein), and 600 grams of water. The food ingredients of this preparation figure out about as follows:

Casein .				2.6 pc	er cent.
Whey proteins				0.4	4.6
Fats				2.6	4.1
Carbohydrates				3.5	6.6
Salts				0.4	**
Water				90.5	4.4

Albumin-cream-milk contains a little less casein and a little more sugar than Finkelstein's albumin-milk. Its caloric value is about 500 calories per liter; Feer says 600, but I cannot see how he obtains this figure. This preparation cannot only be employed like albumin-milk for sick babies, but, like it, seems of some value as a regular article of infant diet. Feer,

however, saw scurvy set in after three months' usc in one case and severe rickets in a few others, so that this modification must also be employed cautiously and excessive sterilization avoided.

ADDITION OF FAT.

Top-milk.—The simplest, and therefore the universal method of increasing the fat content of the infant's food, is to use cream or top-milk in preparing the desired mixture instead of whole milk. Cream obtained by the centrifugal process is so variable in its fat percentage that it can only be used in a laboratory, where the proportion of fat can be checked off by the Babcock test; for general use the employment of top-milk is more accurate and trustworthy. In making use of top-milk it is necessary to know beforehand the percentage of fat in the whole milk; we have seen that this also varies greatly. We can, however, limit this variability, by confining ourselves strictly to milk from the commoner grades of cattle; we may then be sure that the percentage of fat is not above 4, and possibly not above 3.5.

Taking milk containing 4 per cent. of fat as our standard, and assuming that it has stood in a cool place for six or eight hours, the fat ratios of the different levels are approximately as follows:

Upper Next	two ounces	24 pe	r cent.	fat	upper	4	ounces	20.0	per	cent.	fat
Next	44	10	**		second			8.0			44
Next Next	44	6 2			į						44
Next	44	ĩ	44	**	third	4	••	1.5			

Below this level the ratio of fat is about 0.5 per cent. down to the bottom of the bottle: this is therefore the proportion of fat in skim milk.

From the above data we can deduce the following figures:

Upper	4	ounces		20.0 per	cent.	fat
- 11	6	**		17.0	4.6	4.4
4.4	8	4.6		14.0	4.4	4.4
4.4	10	4.4		12.0	4.4	4.4
**	12	4.4		10.0	4.6	
**	16	44		7.5	4.4	64
44	20	**		6.0	4.6	4 4

We must not forget that milk containing only 3.5 per cent. of fat will give results one-eighth lower and 3 per cent. milk one-fourth lower.

Let us consider the various points that make up what we may call the theory of top-milk feeding. In the first place there is the endeavor to provide ample caloric value; in this respect the method must be regarded as successful. Secondly, there is the idea that casein is not very digestible, and that it is advantageous to give it in a rather low ratio, making up the shortage with fats; this principle we have seen to be fallacious. Thirdly, comes the attempt to present casein and fat in the proportions that occur in human milk; to effect this the milk is diluted until the casein is brought down to the desired percentage and top-milk added to raise the ratio of fats, as well as sugar to bring the carbohydrate proportion to normal. A priori, this should give us a faithful reproduction of human milk; in practice, we have learned that this is not at all the case; the facts and experience have run counter to the

theory. Regardless of the mathematical niceties of this method, a very large proportion of infants do badly on top-milk mixtures.

The numerous disadvantages of top-milk feeding arise from two causes: (1) the inherent defects, in addition to those just mentioned, and (2) the technical errors committed in carrying it out. Among the former group, I may mention that the milk-bacteria tend to rise to the top of the bottle, and are nearly twice as numerous in the uppermost two ounces as lower down (Torrey and Rahe); this necessitates the use of exceptionally pure milk, to begin with. Next comes the impossibility, for many young infants, of digesting the cow-milk fats, if given in the proportion of the fats in human milk. No doubt, some babies, especially in the second half-year, tolerate overloading with cow-milk fats, and some even manage to gain rapidly on an excess of this food; on the other hand, nothing is more clearly established than that most of the failures in infant feeding are due to the giving of high fat percentages, as will be shown more fully later on.

Turning to technical errors, we come to the inaccuracy of top-milk feeding, a fatal defect in a method that rests on a mathematical foundation. We have noted the variability of the fat ratio in cow's milk, the exceedingly high proportions furnished by certain breeds of cattle, and the impossibility of controlling this factor unless we have an extensive laboratory equipment at our command. Extraordinary care must be exercised in top-milk feeding in this regard; the mere fact that we run so close to the limit of safety in giving the least digestible milk ingredient causes the danger of overstepping the line to be ever present.

The second technical error, not absolutely inherent in the method, but committed by all its advocates, is the short feeding interval, which is given in all the tables. I shall endeavor to show, later on, how this defect may be eliminated. It is certainly responsible for some of the failures chargeable to top-milk; many an infant might have tolerated it fairly well, if its stomach and intestine had not been persistently overloaded by too frequent feeding.

I pass over the circumstance, that the top-milk method has never paid any attention to the sugar and salt metabolism; these omissions were excusable, at the time that this type of feeding was proposed, as the possible disturbances caused by these ingredients were imperfectly understood; today we have no right to continue to ignore these factors which so often are of vital importance.

Let us now pass on to a few of the feeding methods, based on the theory of top-milk.

1. Biedert's Mixture.—This method is the parent of those that follow. Biedert took 125 grams (4 ounces) of 10 per cent. cream (the upper third of the bottle) and added thereto 375 grams (12 ounces) of water and 18 grams (0.6 ounce) of milk-sugar. As the infant grew older he added successively 62, 125, 250, and 375 grams (2, 4, 8, and 12 ounces) of milk; this made a total of five formulas, giving the following percentages of food-ingredients:

Formula.	ī.	II.	III.	IV.	V.	
Proteins	0.9	1.2	1.4	1.7	$^{2.0}$	per cent.
Fats .	1.0	1.3	1.6	2.0	2.3	- 11
Milk-sugar	4.8	4.8	4.8	4.8	4.8	4.6
Salts	0.2	0.25	0.3	0.38	0.45	1.6
Calories	320.0	350.0	390.0	440.0	480.0	per liter.

The actual number of calories ingested, Biedert's figures giving the daily ration, is 160 for the first formula and 420 for the fifth. These are the amounts we would now give to newborn and two-months babies respectively; Biedert gave them at much later ages. Perhaps this fact accounted for some of his successes, for he certainly did not run the risk of overfeeding with fats and carbohydrates, while the protein ratio was approximately normal.

2. Meigs' Mixture.—Meigs proceeded in somewhat different fashion. He drew off the upper half of the bottle (7.5 per cent. fat), added to three ounces of this top-milk, three ounces of a 15 per cent. solution of milk-sugar and two ounces of lime water. This mixture he gave throughout infancy, merely increasing the quantity as the child grew older.

Made with ordinary milk (4 per cent. fat), the composition of Meigs' mixture is as follows:

Proteins	1.3 per cent.	
Fats.	2.8 ''	
Milk-sugar	7.3 ''	
Salts	. 0.3 ''	
Water	88.3 "	

Meigs preferred to make his mixture with rich milk, endeavoring to obtain the excessive fat percentage of 4.7, quite feasible, if Jersey milk of the best quality is used, but altogether objectionable, as we have seen. The caloric value of the analysis, given above, is about 550 per liter, rather high for earliest infancy, and too low afterward. Meigs' employment of rich milk might raise the number of calories to 770, which is certainly a little too high. His method involves other errors; the amount of proteins is much too low, that of carbohydrates rather high for the first month or two. It is easy to see that there must have been many failures with his modification.

3. Rotch's Mixtures.—Rotch's method is usually regarded as synonymous with the percentage method, and is often called, par excellence, the American method: we have, however, observed that the percentage idea originated with Biedert; the national appellation is rather derogatory, for its narrow limitation to this side of the Atlantic is by no means flattering, foreign observers having noted some of its defects from the first. Credit must, however, be given Rotch for the recognition of two facts: first, that different children require different food concentrations, and that no single formula or even five formulas, will satisfy these requirements; secondly, he saw more clearly than Biedert, that the tolerance of cow's milk varied enormously according to age. In response to these demands, he drew up tables following Escherich⁴ in seeking to imitate, within a fraction of 1 per cent., the poorer, medium, and richer types of human milk, adapting these variations to the different ages of infants, but reserving, which is important to remember, the right to deviate from this routine in the individual case, when the condition of the infant's digestion and metabolism seemed to call for it.

The result is necessarily intensely artificial, and the artificialities of Rotch's tables have been enormously increased by some of his disciples. I may begin by quoting from Ladd¹ a table approved by Rotch himself, here very slightly abridged.

Formula No.	Age.	Per	centage:	s.	Ounces		Interval	
	8	Proteins.	Fats.	Sugar.	per meal.	of meals.	hours.	
1	Birth	0.5	2.0	5 0	1.0	10	2.0	
II	2 weeks	0.5	2.5	5.5	1 5	10	2 0	
III	3 weeks	0.75	3.0	6.0	2.0	9	2.0	
IV	4 weeks	1.0	3.5	6.5	2.5	8	2.0	
V	6 weeks	1 0	4 0	7.0	3.0	8	2.0	
VI	2 months	1,25	4.0	7.0	3.5	7	2.5	
VII	3 months	1.5	4.0	7.0	4.0	7	2.5	
VIII	4 months	1,5	5.0	7.0	4.5	6	2.5	
IX	5 months	1.75	4.0	7.0	5.5	6	3.0	
X	6 months	2.0	4.0	7.0	6.0	6	3.0	
X1	8 months	2.5	4.0	7.0	7.0	6	3.0	
XII	10 months	3.0	4.0	6.0	8.0	6	3.0	

Formula No.	Milk ounces.	Water ounces.	Milk- sugar ounces.	Total calories.	Calories per kilo.
1	1.5 of upper 8	8.5	0.4	125	36
11	2.5 " 8	12.5	0.75	205	60
111	3.5 " 8	14.5	1.0	295	80
IV	5.0 " 8	15.0	1.25	400	100
V	7.0 " 8	17.0	1.25	500	110
VI	10 0 " 12	14.5	1.25	510	100
V11	Upper 12	16.0	1.25	580	100
VIII	" 12	15.0	1.25	580	90
IX	' 16	17.0	1.5	660	95
X	" 20	16.0	1.5	720	100
X1	" 24	18 0	1.5	780	100
XII	40 of whole milk	8.0	1.0	900	110

The first table gives the food requirements. In the second table I give the method by which they may be met, using top-milk, and adding milk-sugar. I add two columns, showing the caloric value of the total day's feeding, and the number of calories per kilogram of the infant's weight, assuming this to be normal for the age.

In making up these formulas the milk-sugar need not be allowed for in calculating the total volume, as its molecules occupy the interstices of the fluid, increasing the specific gravity, but adding extremely little to its bulk.

A few remarks will indicate how artificial this method is and how difficult to work. The tables that I have given presuppose milk, containing 3.5 per cent. of protein, 4 of fat, and 4.5 of sugar as the raw material for modification. If it is lower in proteins and fats we have to take a slightly larger quantity of the same layer, as indicated in the table, and add a little less sugar and water. It is clear that accurate composition is impossible outside of a well-managed laboratory, where the raw material may be analyzed daily, at least for fat, and the ingredients measured precisely. The milk laboratory has indeed always been the cornerstone of Rotch's4 method, and it cannot be denied that his results were better than anything that had been previously accomplished, though it is impossible to say how much of this was due to his requirement. of a very pure quality of milk, and how much to the percentage system. At any rate, the lapse of time has established at least three points of weakness in the

laboratory. The first is the dependence on assistants who may or may not be reliable; to err is human, and so complicated a procedure, as the filling out of these formulas, calls for a high degree of intelligence and conscientiousness. The second objection lies in the fact, that these laboratories, to realize a profit, must remain limited to the large cities; children living elsewhere, must have the modified milk transported to them, possibly hundreds of miles; it is not uncommon, in summer, to find the shipment spoiled on arrival. an eventuality which either causes illness, through not being noticed, or compels the mother to seek a probably inferior temporary supply at a local dealer's. Last, not least, comes the tendency of the ordinary practitioner to interpret the tables in routine fashion, with his eyes glued to the age column, and a devout faith in the mere figures, that would be admirable if it were not so pathetic. It is, indeed, impossible to use the tables intelligently without a thorough knowledge of infantile digestive pathology, easy enough in the hands of an expert like Rotch, but bristling with difficulties to a physician of only moderate experience.

The milk laboratories employ three elements in making up the prescribed mixtures: (1) a cream containing 16 per cent. of fat; (2) separated milk, from which the fat has been removed by centrifuging; (3) a 20 per cent. solution of milk-sugar. It must be remembered that the first and second of these contain nearly the same proportions of proteins and sugar, approximately those of whole milk. The desired

percentages of the three principal milk ingredients are ordered by the attending physician in the form of a prescription which is filled out as at a pharmacist's. For example, if a mixture with 4 per cent. of fat, 2 per cent. of protein, and 7 per cent. of sugar is prescribed, the laboratory lets one-fourth of the total consist of cream, another fourth of separated milk, which is calculated as containing 4 per cent. of proteins, and another fourth of the milk-sugar solution, the total being made up with water. The result adds up as follows:

	Proteins.	Fats.	Sugar.
250 grams cream	10	40	10 grams
250 grams milk .	10		10 ''
250 grams sugar solu-			
tion			50 ''
		_	_
1000 grams total ,	20	40	70 ''

Dividing by 10 we obtain the percentages.

A practical objection to laboratory milk, for persons of moderate means, is its costliness, the usual charge being a dollar a day. The cost of the raw materials, of the very best quality, is less than twenty cents. Because of the frequent necessity of long-distance transportation, as well as the expense, it became imperative to devise some means of bringing percentage-feeding within reach of the masses. This led Chapin¹ to work out the method called home modification.

Home modification, carried out under the supervision of a physician, who uses, as general guides, two tables more or less like those I have given, solves

the economic problem, and obviates the risk of finding the daily supply spoiled, on arrival. Any intelligent mother or nurse can learn to make up the day's total in an hour or two. The measuring of fluids is best done with a Chapin one-ounce dipper, the milk-sugar may be reckoned at three level tablespoons to the ounce (C. H. Smith). The procedure is as follows: The milk is allowed to stand in a cold place for six or eight hours; the desired standard of top-milk is removed carefully with the dipper, and the required quantity of this similarly measured. This is added to the stated amount of water, in which the allotment of milk-sugar has already been dissolved; milk-sugar is more easily dissolved in hot water. Then the entire quantity is heated, either to the boiling-point for three minutes or to 145° F. (63° C.) for thirty minutes; then rapidly cooled and filled into the desired number of bottles, which are then placed in the ice-chest. I need hardly add that the bottles should be warmed to about 90° F. (32° C.) before giving them to the baby; this is most easily done by standing them for some minutes in very warm water. It is a good rule neither to chill nor to heat the bottles too suddenly, if we wish to avoid the risk of breakage.

Aside from what are now regarded as errors in principle, Rotch's tables harbor another defect. The caloric value of his mixtures Nos. IV and V, intended for the second month of infancy, is rather high for artificial feeding, No. VI drops to about the proper figure, and there is another drop between Nos. VII and VIII that brings the latter a little too low. This

defect is directly connected with the excessively short feeding interval in early infancy, and the attempts to correct this, as the baby grows older. I might say that the short feeding interval mars the whole scheme, and that the very small meals only partially compensate for this error, which, in the opinion of modern pediatrists, vitiates the entire program.

From the very beginning, physicians had no end of trouble with percentage-feeding, in a large proportion of their patients; most of these absolute and relative failures were wrongly attributed to an excess of casein, whereas we now know that the high fat ratios were to blame. These experiences led to a desire to vary the percentage plan according to the individual, so as to give different proportions of the various milk ingredients; this in turn gave rise to large series of new tables. Holt² refers to one ambitious author with 579 formulas, a vivid commentary on the riot of mathematics inherent in the consequential study of Rotch's method. The difficulties encountered, in keeping these masses of figures in mind, or even at hand, led Baner to attempt to draw up a simple series of formulas, by which the desired composition of the food could readily be calculated.

Baner sets out with the premise that milk contains 4 per cent. each of proteins, fats, and sugar; the apparent inaccuracy not being so very important, inasmuch as the fats are stated correctly. The calculations become too difficult if we employ many fractions. He then presents the following equations:

Let Q = the total quantity desired (in ounces).

F = the desired percentage of fats.

S = the desired percentage of sugar.

P = the desired percentage of proteins.

M = the required amount of whole milk.

C = the required amount of cream.

A =the percentage of fat in the cream minus 4:

Then:

(1)
$$C = \frac{Q}{A} \times (F - P)$$
, (2) $M = \frac{Q \times P}{4} - C$, (3) $W = Q - (C + M)$,
(4) $S = \frac{(S - P) \times Q}{100}$

As an illustration, suppose that we desire a quart (32 ounces) mixture containing 2 per cent. of proteins, 3 of fat, and 6 of sugar, using the upper 8 ounces (14 per cent. top-milk) as a base. By interpolating in the above equations we obtain the following:

(1)
$$C = \frac{32}{10} \times (3 - 2) = 3.2$$
 ounces.

(2)
$$M = \frac{32 \times 2}{4} - 3.2 = 12.8$$
 onnces.

(3) W =
$$32 - (3.2 + 12.8) = 16$$
 ounces.

(3) W =
$$32 - (3.2 + 12.8) = 16$$
 ounces.
(4) S = $\frac{(6 - 2) \times 32}{100} = 1.28$ ounces.

This method is, at least, within the power of the ordinary, not mathematically trained mind; the formulas given by other authors are nearly all more complicated, some of them excessively so. Even those of Baner are not easy to memorize, and the calculations entail more labor than is justified by the results.

Ten years ago, percentage-feeding had been adopted,

almost universally, by American pediatrists, though it found little favor abroad, being considered too complicated and artificial; in those days only few dissenting voices were heard on this side of the Atlantic. Since then the tide has turned and percentage-feeding is now sensibly on the wane, though a substitute therefor has not yet been agreed on. The reaction was inevitable, because of the serious inherent defects of the method; it is interesting to see how it has been gradually picked to pieces by the criticism of many leading American authorities.

I have already quoted Holt's² opinion of the mathematical orgies, that have been celebrated by the devotees of percentage-feeding; he does not hesitate to say that the percentage idea itself has been altogether overemphasized. Koplik² considers the method overrefined and essentially artificial, taking no cognizance of the normal variations in human milk, which it seeks to imitate; the laboratories are endeavoring to improve on nature, with inferior materials; he scouts the idea of trying to benefit a sick infant, by giving it a food which is fundamentally unsuitable, yet figured out to an amazing nicety. Jacobi¹ breaks out into open ridicule of what he calls the gospel of top-milk; he also notes that infants thrive on human milk of the most varied composition, whereas they often will not digest the top-milk percentage mixtures, though commanded by the formulas to do so. Chapin,2 always a luke-warm follower of the cult, objects to the rigidity of the scheme, which permits of no individualization; his opinion is of especial value, because no pediatrist has given percentage-feeding a more thorough trial, whereas Jacobi has always regarded it askance. Ladd¹ admits that it is good for healthy babies only, and fails in the presence of a digestive disturbance; this amounts to at least partial condemnation, for some healthy infants will stand almost unlimited abuse of their digestive organs. It is only fair to add, that Rotch himself reminds his critics that he never recommended his system as a hard and fast routine, prescribing such and such formulas for this and that age, but drew up his table only as a general guide; as to this point, there is no doubt that his disciples have outheroded Herod, as invariably happens in a new and promising cause, medical or otherwise.

Still worse is the case of home modification, where irregularities in the percentages are inevitable, because it is practically impossible to determine the composition of the raw material, especially as to its fat content. Rotch² admitted unevenness in the laboratory product, and thought that the results of home modification might be equally reliable—faint praise at best but his recommendation of home modification fails to stand the test. Shortly after his publication, Edsall and Fife showed that the laboratories, with all their faults, produced a far more uniform food, than could be made in the home, for the reason just stated. Presently, Holt³ reported a whole series of disastrous results from inadvertent high fat feeding, because excessively rich milk had been used, under the misapprehension that it was ordinary grade milk. Only a few months later Holt4 again brought up the subject. warning practitioners against the use of Jersey milk; his² still later assertion that this excessive fat feeding is due to a misunderstanding of the formulas cannot apply to Rotch's own tables, which are relatively simple and sufficiently clear. There is no doubt, however, that many of Rotch's disciples have succeeded in losing themselves in their own mathematical mazes. Recently Ladd³ has again come to the rescue by showing that high fat ratios are often tolerated. Voices favoring higher fat percentages are now being heard from Germany, where, among others, Stolte² and Heim and John¹ plead for a richer infant diet. I must mention, however, that they refer to a fat percentage of about 3, whereas the Rotch method calls for 4 per cent., a considerable difference. Now, no one has ever doubted that some infants can dispose of an abundance of fat from the beginning, and most of them after six or nine months; this forms no argument against the exercise of caution in fat-feeding. It is, after all, our main object to avoid failures, not to vaunt successes which might have been achieved with any method of feeding; by this test alone, the high ratio of misadventures, the top-milk gospel has forfeited all claim to reverence.

Another grievous fault of the percentage method, already referred to, is the short feeding interval; the digestive tract is loaded up with fresh and rich food before the preceding meal has been disposed of. If we modify the method by introducing the four-hour interval it will of course be necessary to recast the tables, for the meals that are reduced in number will

have to be increased in bulk. For my part I fail to see any benefit from recasting tables that are based on the misunderstanding of so many fundamental principles of infant-feeding; it seems to me a good opportunity to discard the whole ballast of top-milk and percentages and return to simpler methods that had proved themselves at least as successful, but were laid aside because they did not achieve the impossible, namely, to make of cow's milk an adequate substitute for human milk.

Rotch's tables can be adapted to the four-hour interval by slightly modifying the age column, eutting out Formula VIII as useless, and allowing a night-feeding up to the age of three months. The ratios of the ingredients, given in the second table, remain unchanged; though they will cause errors of small fractions of an ounce, in filling the bottles, these trifling irregularities may be disregarded.

Formula		Ounces	No. of
No.	Age.	per meal.	feedings.
I	At birth	1.75	6
II	2 weeks	2.5	6
III	4 weeks	3.0	6
IV	6 weeks	3.5	6
V	2 months	4.0	6
VI	3 months	5.0	5
VII	4 months	5,5	5
IX	5 months	6.5	5
\mathbf{X}	6 months	7.0	5
XI	8 months	8.0	5
XII	10 months	8.0	5

(The amount of whole milk in formula XII to be reduced 8 ounces.)

4. Gärtner's Fat-milk.—Gärtner's fat-milk is one of the favorite German methods of giving a food, low

in proteins, and fairly rich in fats, the proportions being: proteins, 1.5 per cent., fats, 3.0 per cent., and sugar 6.5 per cent., caloric value about 600 per liter. Gärtner's modification is prepared by diluting the milk to reduce the proportion of casein, centrifuging to increase the fats, and adding sugar. Warmly recommended by Escherich,² this product rapidly became popular in Germany, to be finally largely discredited, for the same reasons that Rotch's method is losing ground here. The reader will note the same unwarranted fear of casein, and the same confidence in the digestibility of the fat; it stands to reason, that the final judgment will also be the same. Baginsky¹ sees in it no advantages whatever over ordinary milk-dilutions.

5. Székely Milk.—Székely set to work to remove the casein thoroughly, and succeeded in doing so by subjecting the milk to the action of carbon dioxid under a pressure of 25 to 30 atmospheres. The residual whey was mixed with half as much of a 10 per cent. top-milk, and 1.5 per cent. of sugar was added. The resulting composition is: protein, 1.7; fat, 3.3; sugar, 6.2; salts, 0.6 per cent.; thus it closely resembles Gärtner's milk. Both Gärtner and Székely milk are a little less rich in fat than Rotch's modification, and less harmful to just that extent. Although Deutsch and Grósz report remarkable successes with Székely milk, its use has not spread widely, because its manufacture requires special and complicated apparatus, and is very costly. Both considerations can be disregarded only if commercial exploitation is the main object in view; they are invariably fatal to the prospect of general adoption by the masses, as these complicated and high-priced foods are not sufficiently superior to more simple modifications to warrant the financial outlay.

6. Niemann's Modification.—Niemann² works along new lines by washing butter with water, to remove the fatty acids, and adding the butter to a skim milk-cereal mixture. The removal of the irritating acids should diminish the injurious effects of the cow-milk fats.

ADDITION OF PROTEIN AND FAT.

Casein-fat-milk.—Heim and John¹ have recognized the error of reducing the casein, and, at the same time, have desired to retain the fats. Their product bears a name which may conveniently be anglicized as casein-fat-milk, the original German name being built up of eleven syllables. Heim and John secure the casein and fat in the same way, as for albumin-milk; they give two formulas for very young and older infants, respectively.

FORMULA A.

Remove the curd from $\frac{2}{3}$ liter of raw whole milk by coagulating with rennet at blood heat; stir the curd into $\frac{2}{3}$ liter of hot water; add $\frac{1}{3}$ liter of hot whole milk and 30 grams of dextrin-maltose. Put the mixture in the ice-chest.

FORMULA B.

Remove the curd from $\frac{1}{2}$ liter of milk; stir into $\frac{1}{2}$ liter of water; add $\frac{1}{2}$ liter of whole milk and 50 grams of dextrin-maltose.

The co	omposition	of	these	two	formulas	is	as	follows:
--------	------------	----	-------	-----	----------	----	----	----------

	Formula A.	Formula B.
Proteins	2.1	3.1 per cent.
Fats	2.4	2.9 "
Carbohydrates.	4.8	7.7 "
Salts	0.3	0.4 ''
Calories .	500.0	700.0 per liter.

The above percentages are those given by the authors. The proteins of formula A seem a little low, and the fat ratios seem to indicate derivation from milk containing only 3.5 per cent. We may regard the above analyses as minimum values.

Casein-fat-milk has the merit of supplying proteins liberally, along with a fat ratio that is not inordinately high. The chief argument against its use is, that we can obtain similar results by slight dilution with the addition of carbohydrates as above. We must note, however, that the product here under discussion is low in whey proteins and salts, lower than could be secured by mere milk dilution. The alteration in the proteins is probably immaterial, but the reduction of the salts is likely to be beneficial; we shall presently see how important a role the salts play in digestive disturbances. The reports of other observers who have tried this food are just beginning to come in; Frank is pleased with his results, though he notes imperfect utilization of both proteins and fats; the latter is, however, an inherent defect in cow-milk feeding, which it is idle to hope to eliminate. Further reports would be very desirable, as we may here be dealing with a valuable modification.

Whey-modified-milk.—E. Schloss² has suggested the following milk modification, which he calls whey-modified-milk; it is put up according to two formulas, which differ so slightly that it will here suffice to give the second. He adds together the following ingredients:

Whole milk	143.0 gra	ms
20 per cent. cream	143.0 "	
Dextrin-maltose .	50 to 70.0 "	
Casein (powdered)	5.0 "	
Potassium carbonate	0.2 "	
Water	714.0 "	

The summary of these constituents is as follows:

Proteins .	1.5 per cent.
Fats	3.4 "
Carbohydrates	6.3 to 8.3 "
Salts	0.4 "
Water	88.4 "
Calories	620 to 700 per liter.

Reports show that this mixture must be used with discretion. K. Meyer shows that it may lead to rickets, but this may be said of any artificial food. Leopold² declares it harmless, but that babies sometimes fail to gain on it who afterward may readily be fattened with ordinary mixtures. This evidently applies to older infants, and his later³ report confirms its greater utility during the first half-year rather than later.

CHAPTER V.

MILK MODIFICATION (CONTINUED).

ADDITION OF CARBOHYDRATES.

Various carbohydrates have been added to cow's milk to bring their ratio up to the standard set by human milk, as well as to make up the additional deficit caused by dilution; their addition constitutes an essential feature of milk modification except where it is necessary to keep down the sugar percentage, which is the case only when we are confronted with the clinical picture of sugar intoxication. We have already discussed those modifications, in which the proportions of other ingredients, as well as the earbohydrates, have been altered, and shall now take up only those methods in which nothing but carbohydrates and water is added to cow's This represents the most ancient method of milk modification, the only one that has held its ground, more or less, throughout the history of pediatries; it is not always the best, in individual cases, but is the one most easily carried out by any moderately intelligent mother or nurse, and the one best adapted to routine employment.

This method can be divided into three sub-headings: (1) the addition of sugar; (2) the addition of dextrin or malt; (3) the addition of starch.

- 1. We have discussed quite fully the digestion and assimilation of the various sugars, and therefore need not repeat those data which are detailed at length in Chapter I; we may, however, add a few clinical notes. All the sugars agree in favoring lactic acid fermentation, though the authorities differ as to which leads in this respect; lactose probably ranks first and cane-sugar last among the forms commonly given. There is quite a general agreement that milksugar is more laxative than cane-sugar, which has a tendency to cause constipation; an excess of lactose causes diarrhea with highly acid evacuations (Talbot and Hill). Calvary and Bendix think that cane-sugar is quite as well adapted to general use as milk-sugar, with the advantage of much greater cheapness and availability. Maltose, by itself, does not show any noteworthy superiority over either of the above; the excellent results, obtained with malt feeding, are certainly not due to the maltose alone.
- 2. Dextrin-feeding is also an ancient procedure; roasted starch has been given to infants from time immemorial, usually mixed with plain milk, and has evidently increased the digestibility of that fluid. Added to watered milk it undoubtedly saved many a baby's life in the dark ages of infant-feeding. Liebig was the first to establish a scientific basis for this article of food, though he was possessed of little real understanding of the action of this substance, which

we ourselves, at this late day, can hardly be said to have explained in its entirety. All malted foods contain dextrin, and there is reason to believe that their value largely depends on their being somewhat complicated; such, at least, is the opinion of Usuki and Stolte, who believe that a mixture of carbohydrates is more slowly absorbed than a pure sugar, and therefore tends to check fermentation in the intestine. Southworth explains the matter more definitely, by attributing the antifermentative action entirely to the dextrin, which is not fermentable as such, but only after it has been split into maltose, a process that takes place only gradually, and in the later stages of digestion.

Southworth³ gives analyses of the different dextrinmaltose preparations that are on the market. The dry preparations, including Soxhlet's Nährzucker, Loeflund's Nährmaltose, and Mead & Johnson's Dextri-maltose, consist of about half dextrin and half maltose; the liquid preparations, including the Maltzyme Co.'s neutral maltose, Loeflund's malt-soup, and Borcherdt's malt-soup contain only from 10 to 15 per cent. of dextrin, and are intended to have boiled starch added before using.

3. The addition of starch is also an ancient procedure. When not dextrinized, as described above, it is invariably boiled; raw starch, as is well known, is almost wholly indigestible. We may add the starch to the milk before boiling, as is usually done in Germany; or we may make up a starch or cereal decoction and add it to the milk, such being the prevailing method in England and America.

Starch-feeding has serious and even fatal effects if overdone, but has been criticized with undue severity, on the theory that young infants cannot split starch. We have shown that the diastatic ferments are present at birth, and Kerley and Campbell have found that some infants can dispose successfully of fairly large amounts of starch, nearly an ounce per day at one month of age, and three ounces at six months. These figures, however, do not apply universally, and it is advisable to keep well below them. Czerny and Keller have pointed out the risks involved in excessive feeding with this carbohydrate; it is apt to cause a form of chronic atrophy, called by these authors Mehlnährschaden, i. e., starch-atrophy, characterized by dryness of the skin and muscular rigidity, but relatively slight loss of weight and only moderate intestinal disturbance. Starch-atrophy finally proves fatal unless the diet is changed; we shall discuss it more fully later. This form of atrophy is specially frequent in Germany, where starchy baby-foods and pap-feeding are very popular with the masses; they are less used in this country, where the malted foods have the preference; starch-atrophy is therefore less frequent here, though by no means unknown.

Starch-feeding, by adding a cereal decoction to milk, is practised here on a very extensive scale, but in a somewhat haphazard way, so that Chapin³ has deemed it necessary to call for a standardization. He gives the following analytical results as to proteins, carbohydrates, and caloric value per

quart	${\bf from}$	the	addition	of	${\tt graduated}$	quantities	of
cereals	3:						

	Barley-meal.			Wh	eat-flo	ur.	Rolled oats.		
To quart of water.	Prot.	CH.	Cal.	Prot.	CH.	Cal.	Prot.	CH.	Cal.
I ounce	0.2	2 1	90	0.3	2.2	100	0.3	1.7	80
2 ounces	0.4	4 2	180	0.7	4.3	200	0.5	3.3	150
4 "	0.8	8.4	360	1.3	8.6	400	1.0	6.7	310
6 "	1.2	12.6	540	2.0	13.0	600	1.6	10.0	460
8 "	1.6	16.7	720	2.6	17.3	800	2.1	13 4	620

The amounts of fat are inconsiderable. The four, six-, and eight-ounce additions can be made only with dextrinized flour, which then forms a thick and very nutritious gruel. Strictly only the one- and two-ounce gruels are under discussion here; by using Robinson's patent barley and groats (oatmeal), the decoction can be made in twenty to thirty minutes, whereas the crude grains must be boiled for several hours, and water added to make up the full quart. The favorite milk diluents, of this class, are barley and oatmeal, one-half or one ounce to the quart; even the former decoction contains about 1 per cent. of carbohydrates and furnishes 40 to 45 calories per quart, a supply that is not altogether negligible.

These thin gruels, despite their very low food value, have nevertheless a wide sphere of usefulness, especially as they are too dilute to arouse any apprehension of causing starch-atrophy. Nearly forty years ago, Jacobi² showed that the cereals comminuted the cascin coagula of cow's milk, so that the curd became

physically similar to that of human milk. He did not share the misapprehension, at that time almost universal, of the essential indigestibility of cow-milk casein, but maintained the trouble was due chiefly, if not solely, to mechanical causes, and was not of a chemical nature, so that reduction of the curd to fine flocculi, instead of massive clots, would remove most if not all difficulties. Recent investigations and experiences have fully justified this position, which is now in a fair way of being universally accepted.

We may now pass on to the several feeding methods that involve the addition of carbohydrates to diluted milk.

Jacobi's Method.—Jacobi² simply dilutes the milk with barley or oatmeal water, using the ounce to the pint decoction, and adds cane-sugar to compensate for the deficit in carbohydrates. He varies the degree of dilution, according to the age of the infant, but studiously avoids the error of laying down any hard and fast rule; the baby's general condition, especially its weight, is the criterion by which the sufficiency of the food is judged. This method is so conservative that the mistake of overfeeding can hardly be made; the defects of the scheme are mostly on the other side, because of the low fat percentage and low caloric value of these mixtures.

Jacobi² boils an ounce of barley in a pint of water, adds a pinch of salt and strains; then he adds half an ounce of sugar, and either half a pint or a pint of boiled milk, according to the infant's age. If we reckon

the amount of foodstuffs in his mixtures we obtain approximately the following results:

	Formula A.	Formula B.		
Proteins	1.5	2.0 per cent.		
Fats	1.3	2.0 "		
Carbohydrates .	6.0	6.0 "		
Salts	0.4	0.5 "		
Water	90.8	89.5 "		
Calories .	420.0	500.0 per liter.		

The caloric value of Formula B can be raised to about 560 per liter by adding another half-ounce of sugar without rendering the proportion of carbohydrates excessive; this would make the caloric value of the mixture fairly adequate for the age for which it is intended, namely, the second quarter-year, erring, if anywhere, on the side of safety. We must remember that the caloric control of infant-feeding was unknown forty years ago; Jacobi's rather good results were entirely the fruit of clinical experience.

When the barley water and the low fat ratio cause constipation, which happens very commonly with this diet, oatmeal water, similarly prepared, may be substituted for the former; sometimes there is advantage in giving a mixture of barley and oatmeal. The slightly laxative properties of oatmeal have already been referred to.

In 1876 the short feeding interval was still unquestioned, so that Formula A was divided into eight three-ounce portions, and Formula B into six parts of five and a half ounces each. After the age of six months, Jacobi adds meat broths; then follows a gradual transition to whole milk, through progressively

stronger dilutions, with the addition of bread-crusts and meat-juice after ten months.

The sugar that Jacobi adds to the milk is always cane-sugar. When his method was devised, milk-sugar was hardly obtainable, and Jacobi was quite confident that there was little to choose between these two carbohydrates; his standpoint has been fully vindicated by the recent researches, previously noted.

When Jacobi introduced his feeding method he did so in response to a crying need, for conditions at that time were chaotic, with a prevalence of starch-feeding which, combined with the impure milk, alone obtainable in those days, involved an infant mortality that was simply frightful. While this plan represents a decided reform, and some of its principles hold good to this day, it cannot be denied that both the above formulas lean somewhat toward underfeeding. It has been pretty well settled that the infant after the first few months should get a food-supply equal to 90 or 100 calories per kilogram of weight if it is to gain steadily and normally. These formulas give about twenty calories less, even if the full amount of carbohydrates is extracted from the cereals, which is a matter of some uncertainty. It was largely the recognition of this deficiency, soon shown in practical feeding, that paved the way for the introduction of the top-milk method, which has the tendency to err in the opposite direction. We have seen that if the former is the more trying to the patience of the attendant, who wishes to see rapid progress, the latter is attended with serious dangers that seem to me to outweigh its advantages.

I have always been a faithful adherent of the essential 'features of Jacobi's methods, and have, for a number of years, for my private and hospital use, employed a table that combines the above principles with a slightly higher caloric value, and adapts them to the four-hour feeding interval. In view of the low fat percentages I deem it advisable to feed six times a day during the first few months, which may be accomplished by introducing a night-feeding, or shortening the interval to three hours and a half. Whereas I consider night-feeding absolutely unnecessary, when an infant is nursed, it often becomes unavoidable with artificial feeding, when the fat ratios must be kept low. As a diluent I use weak barley or oatmeal water, half an ounce to the quart, which furnishes about 1 per cent. of starch and an inappreciable amount of protein.

Formula No.	Age.	Number of feedings.	Ounces per meal.	Ounces of milk.	Ounces of sugar.	Ounces of barley water.	Calories per kilo.
I	Birth	6	1.5	1.5	0.25 (1)	7 5	25
11	1 week	6	2.0	3.0	$0.375 \left(\frac{3}{8}\right)$	9.0	45
111	2 weeks	6	2.5	5.0	$0.5 (\frac{1}{2})$	10.0	55
IV	1 month	6	3.0	7.0	$0.75 \left(\frac{3}{4}\right)$	11.0	65
V	2 months	6	4.0	11.0	1.0	13.0	75
Vl	3 months	6	5.0	15 0	$1.25(1\frac{1}{4})$	15.0	85
VII	4 months	5	6.0	18.0	1 25	12.0	85
VIII	5 months	5	7.0	22 0	1.25	13.0	90
IX	6 months	5	8.0	26 0	1 25	14.0	95
X	8 months	5	8.0	30 0	1 25	10.0	100
XI	10 months	5	8.0	34.0	1.0	6.0	100

In the above table the age column is to be employed only as a general guide, to be departed from

freely as the requirements of the individual infant warrant. Either cane-sugar or milk-sugar may be used; I prefer the latter, but the former is cheaper, and possibly more convenient for general use, while not notably inferior.

It will be seen that the quantity per meal, while somewhat larger than noted in Rotch's tables, is hardly up to that ingested by the nursing infant. The caloric calculation is so precise as to appear artificial, and I frankly plead guilty to the charge, claiming, however, that of all artificialities accurate estimation and apportioning of food value are comparatively pardonable.

The percentages in these formulas are as follows:

			Carbo-	
Number.	Protein.	Fat.	hydrates.	Salts.
I	0.7	0.7	4.5	0.13
II	1.0	1.0	5.1	0.19
III	1.2	1.3	5.7	0.25
IV	1.4	1.5	6.8	0.30
V	1.7	1.8	7.0	0.34
VI	1.8	$^{2.0}$	7.2	0.38
VII	2.0	2.3	7.4	0.43
VIII	2.2	$^{2.5}$	7.1	0.48
IX	2.4	$^{2.7}$	6.6	0.52
X	2.7	3.0	7.0	0.57
XI	3.0	3.4	6.6	0.63

The reader will see that the fat ratios are not inordinately high and that the proteins are ample; the salt percentages are low during the first months, the dangerous period. The number of formulas is subject to criticism, being not much smaller than is advocated by the more moderate adherents of the percentage system. I have given so many, largely to satisfy an apparent demand; numbers VI and VIII may readily be dispensed with, and a ten-months infant can often be put on whole milk with perfect safety.

Pfaundler's Method.—Other moves may be recorded in the direction of greater simplicity. As an example, I give a proposition made by Pfaundler.¹ He mixes one-tenth of the body weight of milk with one one-hundredth of the body weight of sugar, and dilutes with water up to one liter; the total is divided into five meals, and the infant allowed to take as much as it chooses. This scheme would work out about as follows:

	po		Js.	P	ge.			
${f Age}$.	Weight, kilos.	Milk liter.	Sugar, grams.	Proteins. Fat.		Carbo- hydrates.	Calories per kilo.	
1 month .	4.0	0.4	40	1.4	1.6	5.8	108	
3 months	6.0	0.6	60	2.1	2 4	8.7	108	
6 months	7.5	0.75	75	2.6	3.0	10.9	108	

Pfaundler's plan is open to several criticisms. In the first place there is an excess of calories, in other words, an excess of food. In earliest infancy this is automatically corrected by the circumstance that the infant will not take more than half or two-thirds of the allotted quantity; later on, when it empties the bottles, it certainly obtains an excess of carbohydrates, the figure for the age of six months being so high as to involve some risk of sugar intoxication. The percentages of proteins and fats are about right. Pfaundler gives malt, by preference, in early infancy, which we shall presently recognize as a good feature; his

recommendation of large proportions of starch, onethird or more of the large total of carbohydrates, in later infancy, must be regarded as an error, inviting the clinical picture of starch-atrophy, as the quantity, recommended by him, runs close to the limit of infantile starch tolerance.

This scheme, furthermore, is too much on the roughand-ready order; it is practically admitted to be such by the author, who wishes to make things easy for the practitioner, in so many words, and evidently desires to achieve a rapid gain in weight, so that all parties will be satisfied. Considering these aspects of the case, this method is not likely to commend itself to the careful pediatrist, and I cannot find any evidence in the literature that it is likely to become popular; it seems to be decidedly a step backward.

Keller's Method.—Keller's³ method of milk modification, devised by him for the feeding of sick infants, but by no means restricted to this narrow field, is essentially a revival of Liebig's, but modernized, and most ingeniously adapted to infants, who have difficulties in digesting the fats of cow's milk. The basis of his plan lies in the employment of what he calls malt-soup, a preparation now put on the market by several manufacturers. Malt-soup, made along the lines laid down by Liebig, contains about 55 per cent. of maltose, 15 of dextrin, and 1.1 of potassium carbonate, besides active diastase. Keller's mixture is made as follows: An ounce and a half (50 grams) of wheat flour are thoroughly stirred into eleven ounces (\frac{1}{3} liter) of milk and strained; then three ounces (100

grams) of malt-soup are added to twenty-two ounces ($\frac{2}{3}$ liter) of water at 122° F. (50° C.); the two mixtures are then thoroughly combined, during which process a part at least of the wheat starch is dextrinized; then the whole is boiled for three or four minutes, to sterilize it and destroy the diastase. The resulting compound presents the following analysis:

$ \text{Proteins} \left\{ \begin{array}{l} \text{milk .} \\ \text{vegetable} \end{array} \right. $	1.1 per cent.
Proteins vegetable	0.7 "
Fats.	1.2 "
Carbohydrates.	11.4 "
Salts	0.4 ''
Water	85.2 "

Its caloric value is about 630 per liter, 19 per ounce. Some points in this analysis are worth discussing more in detail. The vegetable protein derived from the flour cannot be regarded as quite equivalent to the milk protein, as its digestibility is somewhat inferior. The ratio of potash is high, about half of the total salts, owing to the potassium carbonate content of the malt-soup; this is added for the purpose of combating the acidosis that is always more or less present when the fat metabolism is impaired, this last being the usual indication for feeding with Keller's modification. The fat ratio is kept exceedingly low to meet the same indication, and the high carbohydrate content is intended to make up for the deficit in fat.

Keller's mixture is of great value in obstinate cases of cow-milk indigestion, partly because of its extremely low fat content, partly because of its alkalinity. Moreover, the high proportion of carbohydrates is presented in as unobjectionable a form as possible, Czerny

and Keller claiming that the tolerance for malt is twice as great as for either milk-sugar or cane-sugar. Without this large percentage of carbohydrates the caloric value of the mixture would be very inadequate.

Babies fed on Keller's mixture usually gain in weight in a very satisfactory way, but the gain consists too exlusively of fat; the other tissues are more or less starved and, if this food is continued for more than two or three months, the low fat and protein ratios are quite sure to lead to rickets. The absorption of the small amount of fat speedily becomes satisfactory; the stools soon lose their soapy character, partly because of the action of the malt (Freund¹), partly on account of the reduction of the calcium waste. The last is reduced by two factors: (1) the alkaline character of the food, potassium being particularly effective in this respect (Dubois and Stolte); and (2) because there is little free fatty acid to bind the calcium. Nevertheless, owing chiefly to the very small supply, less than the normal amounts of both calcium and phosphorus are absorbed, so that the liability to rickets is readily accounted for. Scurvy has also been observed after continued feeding with Keller's modification (Rosenthal).

Through its success in cases of atrophy and fatindigestion, Keller's idea, suitably modified, has led to a wide extension of malt feeding. Observing the easy digestibility of the malt mixture, pediatrists have endeavored to apply the principle later on, when ordinary feeding, with abundant proteins and fats, must again be given. These attempts have turned out most successfully; the addition of malt to the formulas, in place of milk-sugar or cane-sugar, has been wholly beneficial; the restraint on fermentation, and consequent improved fat absorption, caused by the mixture of dextrin and maltose, has been so evident as to lead to a very extensive employment of this substitute for the ordinary sugars; it is likely to become a permanent addition to our means of milk modification.

I make it a rule to give the ordinary formula with dextrin-maltose whenever the usual milk- or canesugar mixtures seem to cause excessive fermentation and colic, or are attended with the evacuation of soap stools. I decidedly prefer this, as a preliminary measure, to going over at once to some very low fat combination, which can only be a temporary makeshift at best. I also find dextrin-maltose an excellent addition to albumin-milk when the first object of that food has been achieved and a gain in weight is desired; in this way I have succeeded in feeding albumin-milk far beyond the period usually advised, with highly gratifying results.

I must not fail to mention that the dextrin-maltose preparations are put up with and without the addition of 2 per cent. of sodium chlorid. It is rarely advisable to add this salt to the infant's food, as sodium chlorid has not by any means the same physiological action as the potassium carbonate added to Keller's malt soup. I regard the malt preparations without salt as decidedly preferable for our purpose.

CALORIC FEEDING.

The disproportion between the difficulties in making up the mixtures described in these two chapters and the results achieved by feeding them has led many pediatrists to cast about for something both simpler and more effective, and some of us profess to have found the philosopher's stone in what they call caloric feeding. Unfortunately for this new movement, it is only too easy to prove that it begs the question, and therefore cannot lay claim to the dignity of being enrolled as one of the many new feeding methods. It is very much as if we were to call the baby-scales a feeding method; in fact, caloric estimation is not even so good a guide, as it merely represents a theory, whereas the scales show results. This is very clearly pointed out by Chapin⁴ and the Heubners, who show that there may be no gain in weight on an apparently ample provision of calories in the food. Koplik² remarks that all our mathematical data fail to allow for the great variations in the utilization of cow's milk even by apparently healthy infants. These facts are indeed so obvious that only a very uncritical observer could ever be misled into adopting a mere physiological theory as an absolute rule in so complicated and difficult a matter as the artificial feeding of infants. Ladd² and Lamb were unquestionably correct in insisting, even in the early days of this propaganda, that the caloric standard should never be elevated to a method, but only utilized as a guide, a check on the existing modes of feeding, but

inferior, even in this respect, to the careful observation of the infant's digestive functions and body weight.

If I have, nevertheless, given in minute detail the caloric value of the various milk-mixtures it has been done for the sake of completeness and to satisfy a popular demand. A reference manual should aim to give every aspect of the subject; this does not by any means imply an indorsement of every idea that is presented. There can be no harm in stating the theoretical food value of a dietary if we bear in mind that the final test of its usefulness lies in the results achieved and not in our anticipations.

We may now pass on to those methods of milk modification which consist in adding foreign substances to cow's milk.

THE ADDITION OF ALKALIES.

This is an old method of modifying milk, and is based on two errors; the first is the belief that human milk is alkaline, whereas it is practically neutral (amphoteric)—faintly acid to phenolphthalein (Southworth⁴). The second error lies in the hasty conclusion that the acidity of cow's milk is harmful; we now know that fairly large amounts of lactic acid may even be beneficial.

The alkalies most often added are lime water, sodium bicarbonate, and sodium citrate. The addition of potassium carbonate has already been discussed under the head of Keller's malt-feeding, and serves a special purpose, apart from the mere desire to alkalize the food; we need not therefore consider that salt further in this connection.

Lime-water is usually added in the proportion of 5 per cent. of the amount of milk, not the total amount of the mixture. The sodium salts are added in the proportion of 1 grain to the ounce of milk, 2 grams to the liter (quart). It will be seen that the addition to the original milk salts is considerable in the latter case, an amount of sodium being added, approximately, equal to that already present. Lime water is so dilute that the addition of calcium to the milk is quite insignificant.

Among the earliest and best studies on this question is a short article by Wright. He found that the addition of small amounts of sodium citrate, say 0.2 per cent., to the milk caused precipitation of a part of the milk calcium and rendered the subsequent coagulation of the casein finer, more like that of human milk. Larger additions of sodium citrate, from 0.5 per cent. upward, entirely inhibited the coagulation of the casein. The same result is obtained by the addition of the same proportion of sodium bicarbonate.

The action of lime water is somewhat different, as it cannot, of course, precipitate calcium. It has, however, a similar action on the casein coagulation, causing it to be finely subdivided, if added in the proportion of 5 per cent., and inhibiting it if 12.5 per cent. are added.

To put it briefly, these alkalies, in the stated proportions, either delay or inhibit the gastric digestion of casein, relegating its disintegration to the intestines. This is agreed upon by all clinicians, but is not so very important after all, because the proteolysis in the stomach is relatively unimportant in any event, compared to the tryptic process in the small intestine. The question naturally arises, what is the real object achieved by adding alkalies to the food? The answer given by Chapin,⁵ Southworth,⁴ and Pisek is: practically none at all, save in those cases, where an alkali is especially indicated. We have seen, however, that potassium carbonate is to be preferred, in meeting this indication, so that it is doubtful if the addition of lime or the sodium salts is ever required. As to the production of a fine casein coagulum, the cereals do this quite as effectively without impairing the digestibility of that protein (Chapin⁵).

By thus disposing of the alkalies as a class we also do away with the claims of sodium citrate, the employment of which recently threatened to become a fad. It came into prominent notice in 1903, being highly recommended by Variot³ and his school, and immediately taken up by Poynton; thence its popularity rapidly extended to this country, very soon leading to the adverse reports cited above.

ADDITION OF FERMENTS.

There have been many attempts made to render milk more digestible by adding ferments. These ferments may be conveniently divided into two groups, the proteolytic and the glycolytic, the employment of which is based, respectively, on two entirely different theories.

1. Proteolytic Ferments.—These are added on the erroneous theory that casein is a frequent cause of indigestion; therefore, to render it more absorbable, pepsin, rennet, and trypsin have been added to the milk or milk-mixtures. Sometimes the milk has been modified by the addition of one of the various peptone preparations that are on the market, or these preparations have been given in water or gruel. The reader must be careful to remember that by adding merely pepsin or rennin, the casein is not peptonized, but only converted into paracasein; to produce true peptonization the action of the pepsin must be prolonged for hours, or trypsin (or pancreatic extract) used. We have already noted that very little protein is rendered absorbable by the gastric secretion alone.

One way of peptonizing milk is to add a portion of the contents of a so-called peptonizing-tube, shake well, and heat to 100° F. (38° C.), for a few minutes; this method is extremely popular in this country, but it remains questionable whether much of the milk protein is really peptonized. Rennet is a popular addition in Europe, largely in the form of a proprietary preparation called pegnin; the reports on this modification of milk vary in their tenor, but mostly agree with Reinach and Oppler in praising it very faintly, and advising against its employment in early infancy, the very period at which it occasionally seems indicated by clinical conditions. The actions of rennet

and trypsin are combined in the German preparation called Backhaus-milk, which has come into very extensive use. As to this also the best authorities are far from enthusiastic, especially as regards very young babies; I cite merely Baginsky¹ and Thiemich² as observers whose opinions cannot fail to carry weight. Philips² reports very inferior results from the addition of 0.2 to 0.5 gram each of pepsin, pankreon (a pancreatic extract), and emulsin to the daily food allowance; this combination had been highly recommended by Siegert.¹ Siegert's² later attempt to discredit Philips' criticism has been finally disposed of by Czerny, who attributes the whole idea of proteolytic ferment-feeding to a misapprehension of the remarks on the digestive ferments by Czerny and Keller; these authors had never meant to imply that any improvement in infantfeeding could be effected along this line.

It remains for us to discuss the direct administration of peptones. Somatose may be regarded as the type of the peptone powders containing 80 per cent. of so-called peptones (mostly albumoses). The liquid peptone (albumose) preparations are quite numerous; they all contain about 15 per cent. of alcohol, without which they would putrefy, and rarely more than 5 per cent. of the product, after which they are named. They thus are really equivalent to fairly strong wines, containing a little albumose, abundant extractives, and usually a little sugar. Their value as infant foods may be readily gauged from their published analyses, and the practitioner will do best by regarding them as alcoholic stimulants, plus a food value less than that of

equal quantities of milk. To this the contained alcohol adds about thirty calories to the ounce, but alcohol is now generally regarded as a specially undesirable caloric vehicle, even in small doses, except perhaps in febrile conditions, and this remark applies most particularly to infants; in spite of its high caloric value, alcohol is in no sense a food, and the real amount of nourishment to be expected from teaspoonfuls or even tablespoonfuls of these so-called peptonoids may be readily conceived as quite trifling.

Recapitulating, the results obtained through the administration of the proteolytic ferments and their products are very far from brilliant, and fortunately they are rarely indicated, except occasionally in fevers, when the last-mentioned group have a certain field of usefulness, which I do not hesitate to attribute chiefly to their content of alcohol. Their employment should never be more than occasional and temporary, and with regard to their medicinal qualities, rather than their ostensible food value.

2. Glycolytic Ferments.—The term glycolytic ferments must here be construed broadly to include glycolytic microörganisms as well as ferments in the modern sense. This is merely a matter of precision, for these methods date back to a time when this distinction was impossible; the inventors of all these processes thought that they were employing ferments as we now understand the term. We have already discussed the most frequently employed ferment of this class in considering malt-feeding; diastase is, however, not properly a glycolytic but an amylolytic ferment; all

the strictly glycolytic ferments that are employed in modifying milk are living microörganisms.

Lactic Acid Fermentation.—Lactic acid is formed from milk-sugar by the metabolic processes of any of the several varieties of the lactic-acid bacillus, the use of which is extremely ancient, having been practised in the Orient since prehistoric times. In western countries milk has been allowed to become acidified spontaneously, in manufacturing butter, though the modern methods of butter-making start the process with a bacillary culture. In the East, artificially acidified milk, produced by transplantation of the bacilli, has always been a popular article of diet.

Much trouble has arisen from the confusion of acidified milk with buttermilk; both are the result of the action of lactic acid bacilli, but their manipulation has been totally different. Acidified milk is still whole milk, and the bacilli are allowed to remain in it alive and active. Buttermilk has had the clotted fats extracted by churning and the residue sterilized by heat. The latter, fully discussed in the preceding chapter, is practically skim milk, containing free lactic acid, but destitute of any ferment activity. The former is a complete food, with intact and vigorous fermentative capacity.

Introduced into this country by Dadirrian, in 1885, as matzoon, the Oriental type of acidified milk steadily gained in popularity, but only as a semimedicinal beverage, until the publications of Metchnikoff started a wave of enthusiasm for this food. Metchnikoff recognized the profound influence of living lactic acid

bacilli on the fermentative processes in the intestinal canal; active formation of lactic acid interferes with the growth of putrefactive bacteria and materially restricts the production of many substances that are both irritating and toxic. We have seen that, in the normal infant, these bodies are, in any case, reduced to a minimum; the intestinal contents of a healthy infant show the action of few germs, except the predominating lactic-acid bacillus. In the presence of certain pathological processes, however, the situation is very different, and the implantation of living acidifying bacilli may often be necessary, or at least useful, in restoring the fermentative processes, in the digestive tract, to the normal type.

Matzoon, also called zoolak or yoghourt, presents the following composition:

Proteins		3.5 per	cent.
Fats.		3.5	4.6
Milk-sugar		3.7	"
Lactic acid		0.9	4.4
Salts		0.7	"
Alcohol		0.1	44
Water .		87.6	"

It thus consists of whole milk, with about a fifth of the milk-sugar converted into lactic acid and a minute and negligible trace of alcohol.

Bacteriologists differentiate several types of lacticacid bacilli, and some discussion has arisen as to which yield the best results. Matzoon is fermented by the variety bulgaricus, but Rotch and Kendall think that the variety acidophilus may be even better. The probability is that there is little choice among them, provided they are in pure culture and uncontaminated with other germs.

Matzoon, as described, is hardly a suitable food for young infants, because it has the defect of ordinary cow's milk, namely, a high percentage of fat. In early life it still remains necessary to dilute the milk, before or after adding the pure culture of lactic-acid bacilli; if we proceed in this manner we obtain a food that has a wide range of usefulness. Thus, Morse and Bowditch found acidified milk far superior to buttermilk in fermentative disturbances; good results are also reported by Dunn.¹ Brady¹ goes into the matter quite deeply; he too notes the great superiority of acidified milk containing live germs. He first boils the milk, to obtain a germ-free soil, then adds the pure bacillus culture and lets the milk stand in a warm room for twenty-four hours, finally modifying with barley water and cane-sugar, as in ordinary formulafeeding. This mixture must be entirely used up within a day, as it cannot be made to keep indefinitely: after the lactic-acid fermentation has reached a certain point, putrefactive bacteria gradually begin to grow in the milk and, in the course of time, gain the upper hand.

Despite these recommendations, the employment of acidified milk has gained ground but slowly, and many authors confess to disappointment. This is largely due to the unreliability of many of the market cultures of the bacilli, especially such as are sold in tablet form; some of these tablets consist entirely or chiefly of dead germs, which of course are utterly worthless for our purpose. The Journal of the American

Medical Association for December 13, 1913, gives a list of such preparations as are active; the liquid cultures are decidedly more trustworthy than the tablets, but, from their very nature, neither will keep alive indefinitely.

Alcoholic Fermentation.—By adding yeast to somewhat diluted milk, and allowing the mixture to stand in a warm place, we obtain the product known in this country as kumyss, which is not the same as Oriental kumyss. The composition of American kumyss is as follows:

Proteins				2.0 pc	er cent.
Fats				1.9	* *
Sugar				3.3	4.6
Alcohol				0.6	11
Acids (free)				0.3	4.6
Salts				0.4	* *
Carbon dioxid				0.4	11
Water				91.1	

Kumyss contains about 430 calories per liter, of which 40 are credited to the contained alcohol. It is therefore a decidedly insufficient food, and has yielded no noteworthy results in infant feeding. It may, however, claim a limited field of usefulness in febrile conditions, especially in older infants, where a moderately low fat and sugar percentage is indicated.

Kefir, or Oriental kumyss, is a product of mixed yeast and lactic-acid fermentation, with the following composition (according to König):

Proteins				2.7 pe	r cent.
Fats.				1.8	4.1
Sugar				4.1	11
Alcohol .				1.1,	1.1
Lactic acid				0.6'	
Salts				0.4	6.6
Water .				89.3	4.4

This kefir has evidently been made from partly skimmed milk. The ratio of lactic acid is often lower than given in the above analysis, and therefore hardly sufficient to influence the intestinal fermentative processes. The alcohol is altogether objectionable. We must also note that kefir is a very variable product and that the percentages of the ingredients are subject to considerable fluctuations, so that the actual food value is a rather uncertain quantity.

Peiser² advises the use of an alkalized kefir in intestinal disorders. He dilutes with water, half and half, and adds 0.1 per cent. of sodium carbonate; the resulting mixture containing the following:

Proteins		1.5 per cent.
Fats		1.7 "
Sugar		1.5 "
Salts .		0.2 "
Lactic acid		0.2 "
Water		94.9 "

The caloric value is only about 280 per liter, a semistarvation diet; most of the reported effect seems to be due to this circumstance, and this product is evidently adapted to only very temporary use.

We must now consider some milk modifications that do not come under any of the stated headings, but which, nevertheless, present some claim to our consideration.

HOMOGENIZED MILK.

Now that the dread of the indigestibility of casein has given way to the well-justified fear of the milk fat, attention has been given to attempts to modify the latter constituent, so as to render it more absorbable. This has led to the manufacture of what is called homogenized milk, in which is presented an endeavor to increase the digestibility of the fats by reducing the size of the milk globules; this is accomplished by nebulizing the milk with a high-pressure spray apparatus. As Chevalier has shown, the globules are thereby not only reduced in size, but are broken up and deprived of their envelopes; the result is a product, that is far more digestible than unaltered milk, but has the disadvantage of spoiling readily.

The employment of homogenized milk spread rapidly throughout Belgium, France, and Switzerland, but destructive criticism was not long in making itself felt. Bernheim-Karrer, Bourdillon, and others presently reported numerous cases of scurvy resulting from this diet, and Chevalier's insistence that this untoward result was due to decomposition of the food did not offset their criticism, since he admitted its poor keeping qualities. Birk³ is one of the few Germans who have tried it, and he has found it not sufficiently useful to compensate for the above-mentioned risk.

BABY FOODS.

We have already discussed the malt and cereal preparations, called infant foods, that are intended only as additions to milk, and as such often serve a very useful purpose. The following remarks apply to such products as are recommended by the manu-

facturers, and unfortunately also by some physicians, as complete infant foods, requiring only the addition of water. I select the well-known article called Nestlé's food as the type; it consists of desiccated milk, baked flour, and 30 per cent. of cane-sugar; more than a third of the total carbohydrate is present in the form of starch. A dilution of 5 ounces of this food with a quart of water yields the following analysis:

Proteins					2.1 per	eent.
Fats.					1.0	
Carbohydra	ates				14.7	
Salts .					0.3	14
Water					81.9	6.6

The caloric value of this mixture is about 750 per liter.

The proteins might appear to be present in fairly ample ratio, but, being largely vegetable proteins, are subject to some discount for imperfect absorption. The fats are so wofully deficient that any prolonged employment of this food is out of the question. The carbohydrates are present in great excess, and the starch content amounts to more than 5 per cent. We cannot conceive of a food more likely, from continued use, to invite the starch-atrophy described by Czerny and Keller; the risk of rickets and scurvy is great, and the former of these may be expected with certainty, if Nestlé's food is given for a few months. We shall presently see that the development of acidosis is greatly favored by the low fat percentage on the one hand and the overloading with unassimilable carbohydrates on the other.

Imperial Granum is an especially bad infant food, being practically pure flour; the recommendation of this food as a substitute for breast-milk cannot be too severely reprehended, and is almost certain to lead to a fatal result from starch-atrophy.

Less objectionable, but still entirely unsuitable for infants, are such products as Horlick's malted This has about the same caloric value as Nestlé's food, a little more protein, still less fat, and the same amount of carbohydrates, but no undextrinized starch. There is less danger of atrophy, but the prospect of acquiring rickets, and especially scurvy, is excellent. We have already noted the serious and fundamental objections to desiccated milk as an infant food, and shall have more to say on this subject later on; for the present it will suffice to remark that the employment of any such food as the regular diet of an infant is an error of the grossest description. These preparations have always been the most fertile source of the hundreds of cases of fat rickets that still throng our dispensaries, and were formerly common in private practice as well; the danger of feeding these products to babies is even enhanced by their ample caloric value. The infants gain in weight regularly and sometimes more rapidly than normal, unless digestive troubles or starch-atrophy set in, as is often the case; their general development does not, however, keep pace with the gain in subcutaneous fat, and the other tissues, especially the muscular and osseous systems, are apt to be in very poor condition.

CHAPTER VI.

INFANT FOODS OTHER THAN MILK.

ASIDE from milk and milk modifications, many other substances and preparations have been suggested and used for feeding infants. In the following pages I shall discuss merely a few of these, selecting such as have been widely employed, or recommended by good authorities.

Beef-juice.—This may be obtained at home by expressing the juice of raw or slightly broiled beef, or may be bought in the shops, ready for use. There are two ways of preparing this food. We may soak a pound of finely chopped beef in half a pint of water, for six to twelve hours, keeping it on ice, and then express the juice by twisting in coarse muslin. The result is about eight ounces of a fluid, containing the following ingredients:

Proteins		3.0 per cent.
Extractives		2.0 ''
Salts .		0.2 "
Water		94.8 "

According to the second method a pound of lightly broiled steak is squeezed in a meat-press; we thereby obtain only about three ounces of a fluid, of similar composition, save for about 1 per cent. additional

extractives and 0.6 per cent. of fat. The former process is evidently the more economical. As foods, both have little value, not more than 4 or 5 calories to the ounce, with the disadvantage of furnishing only proteins and extractives, no carbohydrates, and little or no fat. The so-called extractives are purin bodies, of some little use in stimulating the gastric secretion, but of low food value, and most probably rather harmful in their influence on the general metabolism. Perhaps no foodstuff has been so persistently and unjustifiably overrated as the article here under consideration.

The commercial beef-juice preparations, represented by Valentine's, differ from the above, only in being about three times as concentrated. They contain an enormous excess of salts (about 10 per cent.), which renders them actually poisonous to infants, even when well diluted.

Beef Extract.—Beef extract is simply concentrated beef-juice, with a slightly higher proportion of extractives and a very large amount of salts; the objections made to beef-juice, apply here with even greater force. Feeding with beef extract is not only a method of almost complete starvation, but is more than likely to induce the condition known as salt-fever in a very short time.

Meat Broths.—When well made, of beef, mutton, or chicken, meat broths are exactly equivalent to dilute meat-extracts; when badly made, as often happens, they are nearly pure salt solutions. The remarks made in connection with beef extract apply here equally. The food value of broths may, however,

be materially increased by the addition of cereals, such as barley or rice, and in that case they may sometimes be of use for older infants, if care be taken to give them strained and in small quantities, not exceeding four ounces at a time, nor replacing other more nutritious food.

Broths were formerly highly extolled as infant foods, either in cases of milk-intolerance, or as supplements to milk in the second half of the first year. It is remarkable how the leading pediatrists of the last generation agreed in recommending these feebly nutritious and even harmful foods, and it is gratifying to note that their vogue has passed. They are still casually mentioned in the text-books, but chiefly for the sake of completeness; all the authorities of today give them a very low rating.

Eggs.—Because of its lecithin content and the widespread but exaggerated belief in the value of lecithin as a source of available phosphorus, the yolk of eggs has been recommended by many authors, among whom I may mention Müller² abroad and Stern in this country. McCollum and Davis show the biochemical resemblance between milk-fat and egg-fat, both differing markedly from the ordinary fats. Stern calls attention to the high ratio of fat in the volk of eggs, namely, 48 per cent., claiming that yolk-fat is more digestible than cow-milk fat, but hardly proving his claim. Other authors may be mentioned who do not share this enthusiasm; Vogt, for example, does not consider the yolk of eggs in any way superior to milk; he finds that infants that are fed on it finally cease to gain in weight and become anemic. As to the metabolic value of EGGS 177

lecithin, we need only to refer again to Berg's investigations, mentioned in the first chapter of this book. It is indeed very questionable if the yolk of eggs forms a very important addition to our list of infant foods.

Egg-albumen-water was at one time a favorite food in severe intestinal disturbances. It was long ago reeognized that these cases tolerated a starvation diet but a very short time, a day or two, and a protein solution was even then regarded as a good food to begin with. Holt gives the following recipe for albumenwater: the white of one fresh egg, one-half pint of water, a little salt, one teaspoonful of brandy; shake thoroughly. This mixture would contain about 1.5 per cent. of proteins and 0.8 of alcohol. I always added the white of an egg, beaten up, to a pint of barley water, with a little sugar added, omitting the alcohol; this gave the rather low ratio of 0.8 per cent. of protein. which was more than compensated for by the addition of about 1 per cent. of sugar and the same of cereal starch. The invention of albumin-milk has rendered egg-albumen-water quite superfluous, so that most modern text-books mention it quite casually.

In connection with the feeding of eggs, special interest attaches to a phenomenon long since familiar to observant practitioners, but first explained by O. M. Schloss, namely, the intolerance to egg-albumen exhibited by a considerable number of persons, including infants. There is a good deal of evidence to show that this intolerance is, at least in some cases, of an anaphylactic nature, so that experiments in feeding infants with either the yolk or the white of eggs are

not unlikely to lead to very acute disturbances, in a certain proportion of cases. Lust⁴ observes that the limit of tolerance for the white of eggs by healthy infants is about 60 grams (the white of two eggs) per day, and much lower in the presence of gastro-intestinal disorders; albuminuria sets in much more readily from this food than from milk proteins, so that we have another very good argument against this form of nourishment.

Leguminous Vegetables.—The dried leguminous seeds (peas, beans, and lentils) contain about 25 per cent. of proteins, 2 of fats, 60 of carbohydrates, 3 of salts, and 10 per cent. of water, with the extraordinarily high food value of 115 calories per ounce. A number of attempts have been made to utilize them in infant feeding. Edsall and Miller added a 10 per cent. dextrinized bean-soup to the ordinary milk mixtures in the proportion of one part of bean-soup to three of milk; this was equivalent to adding about 0.7 per cent. of protein and 1.5 of dextrin to the food. This combination seemed to be of some value in infants that failed to get along on the regular formulas; some of the benefit undoubtedly was attributable to the dextrin. the role of the vegetable protein being rather doubtful. These investigations should be followed up, especially along the lines of the protein metabolism; we know that the leguminous proteins are less digestible than those of animal origin, but we have little experience as to the application of this matter to infants, who may be quite incapable of utilizing this sort of food advantageously.

A few years ago, Ruhräh¹ advocated a similar employment of the seeds of the Japanese soy-bean, a related vegetable, which has, however, the marked disadvantage of containing about 15 per cent. of a vegetable oil. Ruhräh¹ makes up a gruel of about 10 per cent. strength, adding 5 per cent. of sugar, if a higher carbohydrate value is desired, for the soy-bean is decidedly lower in carbohydrates than our common kidney bean, containing barely 30 per cent. Attempts to popularize this food have so far met with little success; the fact is that the leguminous seeds are not very easily digested even by healthy adults, and pediatrists are naturally somewhat cautious in giving such a food to young babies. The prejudice against the legumes may be unfounded, but evidence tending to abate it has not yet been furnished; it is therefore likely that further attempts in this direction will be few and far between until we can administer them along well-established physiological lines.

Carrots.—Carrot-soup was so highly recommended for acute digestive disorders by Moro⁴ and others, that Klotz² felt impelled to investigate this food thoroughly. He found that carrot-soup was, to be sure, only rarely rejected by the infant, but this was about all that could be said in its favor. The digestive trouble remained unaffected, and the protein absorption was very poor, as is probably the rule with the vegetable proteins. The really remarkable thing is that anybody should look for good results, in cases of indigestion, by feeding a woody vegetable like the carrot, rich in carbohydrates, but even in this respect

not superior to a thoroughly cooked potato. In my opinion, endeavors of this kind are simply therapeutic aberrations.

Hemp-soup.—I mention hemp-soup, made of hemp-seed, merely because it also has been proposed by good authorities, and has met the same fate as carrot-soup at the same hands (Klotz³). We need not waste additional space on this unpromising subject.

Banana Meal.—Vipond is the author responsible for the recommendation of banana flour as an infant food. His suggestion presents one obvious disadvantage in that banana flour is a rare market product, not in general demand, and therefore almost unobtainable. Furthermore, Vipond's own reports furnish no evidence of any real superiority to the ordinary cereals. It is credited with being somewhat astringent. but this is rather a disadvantage in feeding normal infants, because they are already subject to constipation. In diarrheal conditions, wheat flour and barley meal have proved themselves so satisfactory as intestinal astringents, which by the way are not usually indicated, that the necessity for other farinaceous foods is not clear. Banana meal is therefore, in all probability, an unnecessary addition to our dietetic armamentarium, and its recommendation is merely additional evidence of the general dissatisfaction with the results of artificial feeding. It represents one of the countless endcavors to find something that will agree with delicate or sick babies, whose real requirement is not this or that infant food but the human breast.

WATER 181

Fruits.—There is practically no literature on the subject of giving fruits and fruit-juices to babies; the entire matter seems to have been tabooed by nearly all pediatrists, and, as a general proposition, this attitude is probably correct, for even cooked fruits, with the cellulose separated, so far as this is possible, are rather unsuitable food during the first year. As to fruit-juices this is not quite so certain; they have a well-defined antiscorbutic action, of which we may often avail ourselves, in artificial feeding, especially if we give mixtures that are more or less throughly sterilized. I have made it a rule to give very small doses of orange-juice to babies who had to be raised on a diet that might possibly induce scurvy, and have seen no harm resulting therefrom. When scurvy has actually supervened, orange-juice is one of the best specifics for this disease; it is always given in quite a liberal quantity, and has never been found injurious in the least degree. Oranges and the related fruits of the genus citrus have the great advantage that they may be given raw; other fruits, such as apples and peaches, must first be cooked, and may not be so effectively antiscorbutic in that state, so that it is hardly worth while to suggest their juices as substitutes.

Of other fruits I desire to mention only pineapples, the juice of which contains a proteolytic ferment. This fruit, that is its juice, might therefore be considered when a proteolytic ferment is called for, but we have seen that this is rarely, if ever, the case.

Water.—It is a constantly recurring question whether we should give an infant water in addition to that contained in its regular fluid diet. This question is usually answered affirmatively in an off-hand way, but seems to me to be entitled to careful consideration for the following reasons. In the first place, most of the formulas, as well as the calculations that have been made in regard to breast-feeding, allow for an amount of fluid that satisfies the physiological requirements under ordinary circumstances; secondly, our present feeding methods already fill the infant stomach to its capacity, and that organ is allowed to be empty for only a short time, with the longer feeding interval, and not at all, according to the older methods; thus there is little room left for additional liquids. The third point is that there is no necessity for water beyond the physiological requirement, and an extra supply merely interferes with the normal water metabolism, causing an increased excretion of urine in the first instance, and increased perspiration and, in extreme cases, diarrhea if the kidneys prove insufficient.

The problem therefore narrows itself down to this: additional water should be given when specially indicated; it only remains for us to point out the indications, and these are relatively simple. First comes hot weather; under this condition it is important that the baby should get rid of body heat, through evaporation from its skin surface, and this is managed by means of perspiration. The most effective means of promoting perspiration, when the surface of the body is overheated, is to give water; this should not, however, be done to excess, and usually 5 per cent. of the body weight; *i. e.*, eight ounces for a ten-pound infant,

WATER 183

given in divided doses through the day, will be sufficient; it is best to give the water when the stomach is presumably nearly or quite empty, namely, about three hours after a meal.

Similar physiological demands are made in the presence of fever, and the body temperature can be somewhat reduced by giving water freely. Here we may give larger quantities and more frequently, as the meals in severe febrile conditions are purposely made smaller and more easily digestible, by giving low fat ratios. When the fever is high we need not hesitate to give one-tenth of the body weight in water per day, which would amount to about four ounces every four hours for a baby weighing fifteen pounds.

Of course, water should not be given to babies iced, but it need not be at blood heat; since our main purpose is to lower the temperature it may be given moderately cool, at the temperature of the room in fever, and at about 70° F. (21° C.) in hot summer weather.

CHAPTER VII.

ARTIFICIAL FEEDING.

It would not be astonishing if the reader emerged from the perusal of the last three chapters in a state of mental confusion. More than a dozen methods of feeding have been presented, each with sponsors and detractors; none is adequate to the satisfactory nourishment of more than a bare majority of infants, many are adapted to merely temporary employment, many more are productive of at least as much harm as good. Unfortunately the confusion that is referred to gives an accurate picture of the present situation; a system of artificial feeding that really meets the requirements is still awaiting discovery; the question is, whether human ingenuity will ever prove itself equal to the task. In the mean time there remains only one proper and reliable way to feed a baby, and that is on breast-milk; artificial feeding is, so far, only a makeshift, a trial and vexation in a large percentage of cases, and only tolerably satisfactory when most successful.

In a large proportion of infants, however, artificial feeding is simply unavoidable; it remains for us to do the best we can with the imperfect means at our disposal.

Based on the experience of twenty years with thousands of infants, my own preference is for the simple method of modification, that I learned from Jacobi, amended to fit the longer feeding interval that has been found best suited to the infant's digestion, and somewhat amplified by the addition of more formulas, so as to furnish a convenient table for ready reference. I have given my table on a previous page (152), but regard it as merely adapted to my own purposes; anybody who understands and employs this method can readily draw up an equally good table for his own use. Jacobi's modification, as already stated, errs slightly on the side of safety; there is a little tendency to underfeed the infant, so that it gains in weight a trifle more slowly during the first six months than the standard tables call for; otherwise I have found it superior, in every respect, to top-milk feeding, which is advantageous only with such babies as digest cowmilk fat with unusual ease, but productive of harm to infants whose digestive powers are less efficient. I therefore always begin by diluting whole milk with the proper amount of barley water, adding either cane-sugar or milk-sugar, according to the financial resources of the family; we have seen that the absorbability of these two sugars differs but slightly, though the latter is, perhaps, slightly more laxative, and therefore preferable.

We may regard a bottle-fed infant as thriving if it does not vomit, voids approximately normal stools, does not suffer from colic, and gains in weight. As to the last we must not expect the remarkably uniform

and rapid increase that we are accustomed to see in successfully breast-fed infants; we should be satisfied if the weekly weighings invariably show some gain, even if the rate, in the successive weeks, shows considerable variation. Daily weighing is unnecessary for apparently healthy infants; the figures are sure to be somewhat irregular, since the bowels and bladder may be full on some days and empty on others, even if care is taken to choose the same hour for the daily weight-taking. This unavoidable irregularity amounts to two or three ounces (50 to 100 grams), much more than the normal daily gain in weight, and is therefore a confusing factor that it is wise to eliminate. Even semiweekly weighing is unnecessary, after the first two weeks, unless we have reason to doubt that the baby is making progress, in which case it may be desirable. Too frequent weighing, especially in bottlefed infants, yields such fluctuating results as often to set the parents' nerves on edge, with the eventual risk of a similar reaction on the attending physician.

A very important matter is not to be hasty in changing the formula because of a temporary slowness in gaining weight, so long as the baby seems comfortable and is evidently assimilating its food. Bottle-fed babies may safely be allowed to lag behind slightly, provided their general condition is satisfactory; I cannot insist too strongly that most of the troubles that arise in the course of artificial feeding are due to overfeeding in the attempt to push up the weight.

If the infant's weight is persistently stationary, or even declines slightly, for two weeks, everything else

being normal, the best procedure is to pass on to the next higher formula, which may be done without regard to the age column, which, as previously stated. is only a general guide; in many of these cases it is quite likely that the proteins as well as the fats are insufficient. If the baby still fails to gain, and the feces indicate that an increase of fats is likely to be tolerated, we may replace whole milk with a 24-ounce top-milk (5 per cent. fat), advancing, if necessary, to a 20-ounce top-milk (6 per cent. fat); it is very rarely that we shall be called upon to go beyond this ratio. By this time the baby will be at least six weeks old, when the fat-proportion resulting from the mixture last mentioned will not exceed 2.8 per cent., which is far below the 4 per cent. ratio allowed by Rotch's tables at that age.

I do not necessarily pass on to the next formula at any fixed time, but let myself be guided by the infant's weight, increasing the amount of milk only if the gain is not satisfactory. With the conservatism in fat dosage that is a feature of this method, there is less danger of developing the fat type of rickets than when we give high fat and low proteins to babies who tolerate cow's milk easily, gain rapidly in weight, but do not acquire a proportionate growth of bone and muscle. The appearance of such infants is most deceptive when regarded with an uncritical eye, and they give a false impression of robustness that is quite unjustified.

My table does not provide for giving whole milk, forty ounces per day, until the twelfth month, but many infants anticipate the schedule almost from the beginning and can take whole milk successfully a month or two earlier.

Many, possibly the majority of physicians, still prefer to employ the top-milk percentage method, with its high fat ratios. There is no doubt that this plan has scored thousands of successes, and that such babies as tolerate it often thrive exceedingly well, especially in the second half of the first year. Some of these infants almost rival nurslings in weight and outward appearance, though the practised eye misses a certain rosiness of color and the experienced hand the rubber-like hardness of flesh that is usual even in second-rate breast-fed babies.

If in spite of what has been said the reader has made up his mind to feed with top-milk from the first, I would urge him to take to heart the following words of caution. First, he should on no account give the frequent feedings, originally recommended, but employ the modified table (p. 139), which has been corrected for the four-hour interval; in this way one main source of trouble, namely, overloading, will be effectually guarded against. In the second place, he should make sure that his supply of stock milk is derived from Holstein or grade cows, and does not contain over 4 per cent. of fat: Rotch's method, as it is, touches the limit of safety, and the slightest step over the line is an invitation to disaster. Thirdly, he should watch the infant, and especially its evacuations, with particular care, reducing the fat ratio on the least suspicion of trouble. If these precautions are sedulously observed he will undoubtedly achieve a certain percentage of successes with this method, and also, in my opinion, develop an unwarranted feeling of security, which will receive a rude shock as soon as he encounters one of the many babies who take unkindly to this sort of feeding.

If the practitioner chooses to avail himself of one of the milk-laboratories, now to be found in practically all of our larger cities, the preparation of the formulas is taken out of his hands, and those of the family, and the chief risk involved, is the possibility, already mentioned, of the spoiling of the day's supply in transit. If, however, he prefers home modification, to which any intelligent mother or nurse can be trained, he must inculcate the following special precautions, aside from the one already mentioned, regarding the quality of the stock milk, which should be the best obtainable and low in fats.

Home Modification.—The required apparatus includes an 8-ounce glass graduate, a glass funnel, a Chapin 1-ounce dipper, a two-quart mixing pitcher, feeding-bottles, an ice-box, and a sterilizer. In addition there should be a plentiful supply of rubber nipples, cotton, and bottle-brushes. The bottles should be graduated and cylindrical, and fairly wide mouthed, to facilitate cleaning; the bottles with tubing attached, which defied all attempts at cleaning, have happily almost disappeared from the market. The holes in the nipples should be sufficiently large to permit rapid dropping of the milk, but not free flow in a stream. When not in use the nipples should be kept in a solution of boric acid. Bottles should be washed immediately after feed-

ing, with cold water, followed by a thorough scrubbing with hot soap-suds, using a bottle-brush, then kept filled with boric acid solution, and rinsed with pure water just before refilling. The nipples should also be scrubbed with hot soap-suds, and a clean tooth-brush immediately after feeding.

The whole day's food should be prepared in the morning, according to the physician's formula, taking the precaution of dissolving the sugar in the hot water or cereal decoction, before gradually stirring in the milk. The result is then filled into the proper number of bottles, which are stoppered with cotton, placed in the ice-box, taken out one by one as required, warmed to near blood-heat, and last of all capped with a nipple.

These regulations apply to all the home modifications of milk; the only difference is in the ingredients. As to the more difficult modes of milk modification, I have given the methods of preparation, one by one, in the previous chapters. Of course, the whole milk dilutions, consisting solely of plain milk, cereal water, and sugar, are the most simple of all to prepare; some of the more complicated mixtures, as albumin-milk, have to be demonstrated by the attending physician or a specially instructed nurse.

Let us pass on to the digestive difficulties, encountered in artificial feeding.

Constipation.—Constipation is the bugbear of nearly all artificial methods of feeding, and its minor degrees are often almost unavoidable, being chiefly due to the poor absorption of fat, or the low percentage that has been necessitated by a regard for the infant's safety,

at least during the earlier months. If the constipation is only moderate, with rather hard movements, instead of the normal semisolid evacuations, and the baby seems to suffer no harm or special discomfort, it is often the wisest plan, to leave well enough alone or substitute oatmeal for barley, but many infants do tolerably well, gaining fairly rapidly in weight, in spite of some torpidity of the bowels. If the constipation becomes troublesome, leading to occasional attacks of colic, or to straining at stool, our first step should be the same as recommended for failure to take on weight, namely, to pass on to the next higher formula; similarly, the giving of higher fat percentages should be resorted to cautiously, since the danger involved in high fat feeding, enormously outweighs the mischief caused by moderate constipation.

The physician must ever bear in mind that artificial feeding places the infant in a dilemma. On the one hand the low fat diet, poorly assimilated at that, favors the development of rickets, and in any case interferes with the full muscular growth of the intestinal walls, so that a tendency to costiveness is unavoidable. On the other hand, any attempt to give cow-milk fat in the proportion in which fats are present in human milk involves one of the greatest possible risks to which an infant can be subjected. Confronted with this situation, we do well to be satisfied with a partial success and not aim at the attainment of perfect results.

If the constipation is severe and causes discomfort, and we have pushed the feeding as far as is safe, the best plan, in my experience, is to substitute one of the dextrin-maltose mixtures for milk-sugar or cane-sugar. It is not that the malt is, in itself, especially laxative, but we have seen that it acts as a check on abnormal fermentation, such as is apt to occur when the bowels are costive. It must be remembered that the discomfort that attends constipation is due to fermentation and distention by its gaseous products, not to the mere presence of somewhat less fluid and more slowly moving intestinal contents. The hard feces of constipation become a feature only in the large intestine, whereas the fermentative processes begin much higher up. Many an infant may be relieved of the more distressing symptoms of constipation by giving malt; I may add that malt-feeding can be kept up throughout infancy if necessary, and that malt is no more likely to lead to rickets than either cane-sugar or milk-sugar. We shall go into the etiology of rickets more fully later; at this point it will suffice to say that it is in no way related to sugar metabolism. The danger of causing rickets, that is associated with Keller's original maltsoup mixture, lies in its low food value, especially in its exceedingly low protein and fat percentages; the babies who are fed on this compound too long are simply starved into rickets. The reader will understand, by what has gone before, that feeding on Keller's malt-soup, and substituting malt for milk-sugar in an ordinary formula, are two very different things, and intended for very different purposes.

While we may, in desperate cases, occasionally resort to glycerin suppositories or soap-suds enemas to relieve infant constipation, any regular resort to

such methods is an error so long as our dietetic resources have not been exhausted. Magnesium peroxid, in the form of milk of magnesia, is given extensively, in fact too often; I have seen as many failures as successes with this salt, and do not agree with Donath, who says that a daily dose of 1 gram of magnesium peroxid may be relied on to be effective, without diarrhea and without habituation. A still greater blunder is the resort to the ordinary purgatives, especially rhubarb, with its constipating after-effect. Intractable constipation in an infant is a classical symptom of rickets, and if dietetic measures fail, we may generally rest assured that a rachitic condition is already established. The best proof of this relationship is furnished by the experience that a vigorous antirachitic course of treatment is more effective, in these cases of chronic constination, than any merely symptomatic measures.

Colic.—Intestinal colic is usually a symptom of some severe intestinal disturbance, but sometimes, as already indicated, may attend mere constipation; in the latter event it is only occasional, and does not call for any special method of treatment.

Vomiting.—The young infant vomits easily and without effort; surplus food readily overflows through the feebly closed cardiac orifice of the stomach. Partial vomiting, i. e., the regurgitation of only a small portion of the ingesta, is popularly designated as "spitting." Spitting is a frequent and annoying symptom in artificial feeding, but has become far less common since the introduction of the four-hour feeding interval; it is

simply the result of overloading the stomach, and an almost unavoidable consequence of allowing too short a pause between feedings. The treatment of this condition is indicated by the above account of its etiology.

Apart from mere spitting, vomiting is always a symptom of some seriousness in a bottle-fed child; if it is persistent and uncontrollable, beginning about two weeks after birth, it is a characteristic feature of the affections known as pyloric stenosis and pylorospasm. Excluding these affections it is a sign that cow's milk, at least as given, does not agree with the infant; the case is one of digestive disturbance and must be treated accordingly, unless the vomiting occurs at long intervals. In many cases vomiting may be checked by going back one formula, or abandoning top-milk, if that has been given, in favor of properly diluted whole milk.

Occasional vomiting is one of the common and unpleasant concomitants of top-milk feeding; and has been one of the chief factors in impairing the credit of this method; as the top-milk percentage system, as usually carried out, combines both the evils of too frequent feeding and too high fat ratios, it is easily understood why vomiting so often is associated with this mode of raising infants; with careful avoidance of the two mentioned errors, this distressing symptom will nearly always be controllable.

Diarrhea.—In breast-fed infants, two, three, or even more green and loose evacuations, although they may contain whitish particles of undigested fat, are of no very great significance; in bottle-fed infants, on the other hand, they should always be regarded as danger signals. Even a mild attack of diarrhea is usually a symptom of fat-dyspepsia which, taken in time, may often be promptly checked. I treat such cases by giving a single purgative dose of castor oil, cutting off all food for twenty-four hours, and then starting in with a very low fat formula, often using skim milk or buttermilk to begin with. These attacks of diarrhea in otherwise thriving infants are usually due to attempts to give high fat mixtures, and therefore specially common in top-milk feeding. It must not, however, be forgotten that the presence of fever, from any cause, is very apt to be attended with intolerance of fats, without any demonstrable error in the direction of overfeeding; in these cases it is also well to go over to a low fat regimen until the febrile disturbance has abated.

Should these slight diarrheal attacks tend to recur, or should the infant have the normal number of stools, namely, one or two daily, but of diarrheal character, the wisest plan is to proceed at once to malt feeding, substituting a dextrin-maltose mixture for the added sugar. We have seen that malt decidedly favors fat absorption, and it is remarkable to see how rapidly the tendency to diarrhea often ceases on a diet containing a low percentage of fat and sweetened with malt.

If the abnormal stools, just described, are disregarded one of two events is likely to take place: the baby either goes over into the condition that the Germans call *Milchnührschaden* (milk-atrophy), to be

discussed in detail later, or develops true fat-diarrhea, already referred to as a serious symptom. I have before described the yellow and oily fat stools, nearly normal in color, very fluid, and leaving greasy spots on linen and paper. In the light of our present knowledge the development of fat-diarrhea is nearly inexcusable, because we have become fully acquainted with its origin; it can occur only if we ignore the warnings given by the less grave anomalies of the feces, and persist in giving fats, in the ratio of 3 or 4 per cent., merely because certain authorities still recommend this mode of feeding as a perfectly safe routine diet.

Soap Stools.—These have already been described; they are often an almost unavoidable accompaniment of bottle-feeding, and are associated with more or less constipation. Soap stools are to be regarded by the attendant in very much the same way as the diarrheal evacuations previously mentioned; they represent a milder type of fat-indigestion, unattended by fermentation or irritation, because here the calcium soaps play a more prominent role than the free fatty acids, which are the prime factors in diarrhea. The seriousness of the soap stools is therefore perhaps less imminent, but they may also pass on, rather suddenly, to fat-diarrhea, so that we should do our best to get rid of them by readjusting the diet. In accordance with the similar etiology the same treatment as for diarrhea applies to soap stools; by going back to a moderately low fat mixture with malt we shall succeed in securing more nearly normal evacuations in a vast majority of these cases.

Viewing the difficulties of artificial feeding in their entirety, two clinical landmarks stand out prominently. In the first place we observe that the feces are a better guide than even the body weight in gauging the success or failure of any method of artificial feeding. We may sometimes disregard the baby-scales for a week or two, but we cannot, with safety, ignore the condition of the bowel-movements for a single day. Whoever wishes to succeed in artificial feeding must comprehend the terminal stage of his milk-mixtures as thoroughly as he understands their original preparation. In the second place we find that there is only one reliable remedy when every attempt at formula feeding seems to go wrong, and that is breast-milk. It is true that, under many circumstances, especially in this country, the remedy is practically unobtainable; this does not lessen its value, and the lack of it very often puts the infant in the greatest jeopardy.

There is a certain percentage of infants who cannot obtain breast-milk for various reasons, and must therefore be nourished artificially, who keep the parents and medical attendant in a constant state of anxiety, because they will not thrive on any form of milk-modification. During the first three to six months they gain only fitfully, often only to lose their hardly gotten gains, because they do not tolerate a mixture that is sufficiently high in food value to more than just keep them alive; of course, their very lives are continually in the balance, and the difficulties of the physician can be readily understood. There is, however, one encouraging side to the situation; at some

ill-defined period, sometimes in the third month, sometimes not until later, the baby suddenly begins to absorb and assimilate its food, and the last formula, as well as the last consultant, gets the credit of what is largely a coincidence. Some of these cases, unfortunately, fall victims to their food-intolerance or to some intercurrent affection that would be overcome by a normal infant. The mortality among infants, from infectious and other febrile diseases, depends to an enormous extent on whether they have been breastfed, successfully bottle-fed, or belong to the group of feeding-failures. These last babies represent an almost irreducible minimum, of whom a certain illdefined percentage has barely an equal chance of survival and for whom the most ingenious plan of artificial feeding will remain a physiological misfit.

Feeding During the Second Half-year.—During the first half-year, as we have seen, it is a good rule to allow all errors in feeding to be on the side of safety and to permit a slight degree of underfeeding rather than take any risks. There is no occasion for worry if the baby is a pound or two underweight, provided it shows no signs of actual illness; Birk⁴ has shown that the body length suffers impairment only in very severe malnutrition, but that a small deficiency in weight, in the earlier months, may easily be made up during the second half-year. After the age of about six months many infants, who previously had difficulty in utilizing cow's milk begin to tolerate and assimilate that food quite well; if the weight at that age is not quite satisfactory, and the baby's condition

and especially the stools, make it evident that a richer food is desirable, we may at once proceed to run up the fats.

At six months of age my table calls for a mixture consisting about two-thirds of milk and one-third of barley water, yielding a protein percentage of 2.2 and a fat percentage of 2.6. If we take the allotted 26 ounces of milk from the top of the bottle, rejecting the lowest 6 ounces, we obtain a 4.8 per cent. milk which will, on dilution, give a fat ratio of 3.2 per cent., not excessively high for that age, but giving a daily addition of 1.4 ounce of fat, 70 calories per day, and about 10 calories per kilogram of weight. This increase will generally suffice to convert a slow gain into a rapid one without incurring much risk. As already observed, it is during the second half-year that the top-milk percentage method has scored its great triumphs; some of its features may very well be borrowed at this period, with profit to the infant, and no hazard of consequence, if we are careful to continue close observation in each individual case.

MIXED FEEDING.

When the two-hour feeding interval reigned supreme, it often happened that a mother could not supply a sufficient quantity of milk with such frequency; this often led to what has been aptly called mixed feeding, the scanty ration of mother's milk being eked out with a suitable cow-milk mixture. Since the adoption of the four-hour interval this trouble has

become less common, but is still by no means rare. All authorities agree that mixed feeding, while inferior to breast-feeding by itself, has many advantages over exclusively artificial alimentation. The infant obtains, at any rate, a certain quantity of easily digestible food, so that the supplementary bottle-feeding may be given with a very low fat percentage without incurring the risk of fat starvation, the mother furnishing a fairly adequate amount of this milk ingredient.

Voix discusses the three possible methods of applying mixed feeding: (1) we may alternate between the breast and the bottle; (2) we may give the bottle in the daytime and the breast at night, or *vicc versa*; (3) we may supplement each nursing with a small quantity of the artificial food. He prefers the third method, which is often easily carried out, by adhering to the four-hour feeding interval.

Périer looks at the question from the sociological point of view, and gives four reasons for mixed feeding:
(1) if the breast-milk is insufficient; (2) if the mother has to earn her living, in which case the bottle will have to be given once or twice during the middle of the day; (3) in raising twins, for obvious reasons; (4) as a transition to weaning. The third of these reasons is also dwelt on by Eustache; it will be recalled that we cannot, as a rule, reckon on a woman's yielding more than one or one and a half liters (quarts) of milk per day (Thiemich¹); this quantity is pretty certain to be insufficient for the nursing of twins, after the first few months, so that supplementary feeding is quite likely to be called for as the twins grow older.

Mixed feeding has been most assiduously studied in France, and is inculcated at the French milk-stations whenever possible. There is surprisingly little literature on the subject in other languages, and it is evident that this method has not received the universal attention that it deserves. The physicians of other nationalities are too easily discouraged and go over to exclusive bottle-feeding much too readily.

In my own experience I have had the best results with Voix's first method. It is very often possible for the mother to nurse her baby three times a day, namely, at 6 A.M. and 2 and 10 P.M.; this will necessitate only two artificial feedings, at 10 A.M. and 6 P.M. respectively. This plan is specially applicable to those cases where the mother's mammary gland is of the virginal type, and secretes an insufficient quantity of milk the quality of which is quite satisfactory. In these cases I have obtained a number of excellent results, lasting for a number of months, and tiding the infants nicely over the critical early period.

THE FEEDING OF PREMATURE INFANTS.

On this subject there is a considerable amount of recent literature, from which we may draw freely. No infants are in more urgent need of breast-feeding than such as are born before the full term; yet these very babies are most apt to be deprived of their natural food. Several reasons combine to produce this unfortunate state of affairs. In the first place the mother's breasts are not supposed to furnish milk at seven and a half or eight months, and, as a matter of fact, usually fail to do so; in the second place, the premature infant's power of suction is feeble, so that the most potent factor in promoting the free flow of milk, namely, the drawing of a vigorous baby, is lacking. In exceptional cases it is possible to obtain the milk of another woman for the premature infant; in this happy event our chance of raising it successfully is immeasurably improved. As a rule, however, we are forced to resort to artificial feeding from the outset, and the difficulties involved in this procedure are among the greatest that fall within the province of the pediatrist.

Infants weighing over 2 kilograms (4.5 pounds) at birth run only the risks proportionate to their backward development; sometimes their mothers are able to nurse them, in other cases artificial feeding is finally successful, so that the mortality is not so very much larger than in babies of the normal weight of three to three and a half kilos (6.5 to .7.5 pounds); it is the babies under the 2 kilogram limit that here specially concern us.

The caloric requirements of premature infants are relatively very great, because of the proportionally large body surface, and consequent excessive radiation of heat. Unless we supply artificial warmth we cannot raise these babies at all; they use up all their available energy in keeping up the body temperature, but do not even succeed well in this; their temperature falls below normal, and they soon die of inanition. Birk⁵ is undoubtedly mistaken when he puts the energy-quotient of these premature infants on a level with

that of full-term babies; Samelson¹ sets it at 115 to 150 calories per kilo, J. H. Hess at 100 to 130 for babies weighing over 3.5 pounds, 115 to 170 for still smaller infants. It is evident that the low diet that is usually given to full-term infants during the earliest weeks of life will not suffice for these tiny morsels of humanity; on the other hand, the dangers of overfeeding them are aggravated by the poor development of their digestive organs. Oppenheimer advises us to go ahead boldly, starting with at least 90 calories per kilo daily, and advancing to 120 calories if the smaller amount has been well tolerated. Morse² and J. H. Hess agree in saying that the infant will not grow on less than 120 to 140 calories, the latter figure being admittedly on the absolute danger line. We have seen that feeding so high as this is unsafe even for fully developed babies when several months of age; it is palpably hazardous under the conditions here being considered, as has been admitted, and even insisted on, by Cramer² and Samelson.1

Our dilemma is only too apparent. Ladd⁵ and Morse³ report poor results; a gain of more than two ounces per week is regarded as uncommonly good; it usually averages lower for a number of weeks, even in cases that finally come out successfully. Birk⁵ considers buttermilk superior to whole milk, because the digestion of fat is specially poor in these infants; Litzenberg urges the four-hour interval for premature babies also. It is difficult to see how the last two recommendations can be made compatible with the high caloric values that are usually demanded; to

my mind these propositions involve a mathematical impossibility.

Personally, I do not even attempt to feed up to the high caloric standards referred to above; nevertheless, I feel that I am trespassing over the danger-line in every case. To babies weighing over 1500 grams (3.5 pounds) I give my third formula, in 1.5-ounce portions, every three hours, day and night; the caloric total is about 140, say 70 to 90 per kilo, and at that I am aware that I am counting on a power of assimilation rather above the average. Still smaller infants get one-ounce portions of the same mixture at the same intervals, totalling about 100 calories per day, and about the same per kilo, as for the larger babies. Here the chances are poor, at best. Millon finds that only 36 per cent., of the babies under 1500 grams, survive; Ladd⁵ gives the still lower proportion of 28 per cent. From 1500 to 2000 grams (3.5 to 4.5 pounds), the outlook is considerably better, the respective figures of these two authors being 75 and 56 per cent. The limit of viability may be set at 1200 grams (2) pounds and 10 ounces), though we may sometimes succeed in saving the life of a still smaller infant.

The conservation of the premature infant's body heat, by means of a warm room and warm water bottles, is of course an indispensable requirement if we are to be at all successful. The electric pad, if skilfully managed, so as not to be overheated, is often a very valuable aid. The incubator, once so universally popular, is gradually falling into disfavor; it calls for special training in its use and an immense

amount of supervision, yet remains a rather dangerous apparatus in spite of the best attention and care. In a manual solely devoted to the feeding of infants there is no place for lengthy discussion of the merits and demerits of the incubator; it will suffice to point out the significant circumstance that many leading clinics, such as Langstein's, which formerly employed the incubator, have recently given it up (Rott).

CHAPTER VIII.

DISORDERS OF DIGESTION.

The disorders of the digestion, in infants, may be divided according to their etiology into three groups:

- I. Physical disorders.
- II. Alimentary disorders.
- III. Infectious disorders.

We shall begin with a consideration of such digestive disorders as have a purely physical basis; they agree in presenting a normal condition of the gastro-intestinal mucons membrane, and in this respect differ radically from the others. They are variously either functional or organic, and present characteristic clinical pictures that separate them sharply from the second and third groups, whereas among themselves the differential diagnosis may often be quite difficult.

PHYSICAL DISORDERS OF DIGESTION.

Pyloric Stenosis.—Congenital hypertrophic pyloric stenosis was first described by Beardsley, but his account was soon forgotten. Its symptomatology was accurately described by Williamson, but this paper also seeured little attention. Hirschsprung was the first one to popularize a knowledge of this affection,

and since his time some hundreds of cases have been reported, chiefly within the last twelve years.

Pyloric stenosis presents a definite anatomical lesion, of which I² gave one of the first detailed descriptions in 1905. The essential feature is an enormous hypertrophy of the muscular coats of the pyloric end of the stomach to fully four times the normal thickness; the enlarged pylorus presents a sausage-shaped mass, projecting into the lumen of the duodenum, its own orifice being narrowed to the diameter of from 2 to 3 mm., and its walls being of almost cartilaginous hardness. In uncomplicated cases the mucous membrane of the organ is absolutely normal.

Symptoms.—The symptomatology is characteristic of obstruction at the lower gastric orifice. There is vomiting of practically all ingested food, at varying intervals after meals; either constipation, or fairly frequent small green and mucoid evacuations, of the type known and described as hunger stools. The child presents the typical picture of progressive emaciation, but no fever or other symptom that would point to an infectious or toxic process.

On examination we find the physical signs of dilatation of the stomach, often with visible peristalsis. Sometimes, but by no means always, we find a palpable tumor in the right epigastric region; the deep situation of the pylorus often interferes with its successful palpation.

The first symptoms of this affection appear at the age of two or three weeks, when the infant, hitherto apparently perfectly well, begins to vomit, though there is no evidence of any error in the diet. The majority of these babies are indeed breastfed, but this is true of babies in general; it merely shows the absence of a dietetic cause. The vomiting soon becomes persistent, and presently very intense and forcible, not by any means resembling regurgitation or "spitting." From this time onward the infant begins to lose in weight, and from having been in a flourishing condition at the age of two or three weeks, rapidly becomes reduced almost to a skeleton.

Unless relieved by treatment the disease continues unchanged, and the infant dies of inanition before the end of the third month. The postmortem findings have already been described.

Before discussing the treatment of pyloric stenosis it will be best to give a brief description of a clinically similar affection, easily confused with it, namely:

Pylorospasm.—Pfaundler² was the first to give a good account of pylorospasm, which was, for some years, confused with organic pyloric stenosis. For some years writers on these subjects were divided into two camps, consisting respectively of those who regarded the clinical picture as due to organic obstruction and those who looked on it as a muscular spasm, with perhaps a secondary development of hypertrophy. Ibrahim³ tried to compromise between these parties by recording his belief that the hypertrophy was the result of the spasms and might disappear spontaneously on their cessation, thus accounting for the undoubted cases of recovery from this group of symptoms. I must mention that Pfaundler² thought that

the enormously enlarged pylorus represented a postmortem contracture, since it was evident that these cases could otherwise be curable only on the basis of an hypothesis such as was later supplied by Ibrahim.³ This is disproved by noting the great enlargement of the pyloric end of the stomach in every direction as well as in total bulk; these large masses of muscle cannot be the product of a mere postmortal spasm.

Koplik,³ a few years later, took the standpoint that has ever since been generally accepted, namely, that there are two forms of pyloric obstruction in infants: An anatomical stenosis, due to hypertrophy of the muscular wall, and a functional occlusion, due to muscular spasm. Whereas knowledge of the former condition has practically stood still since the publication of my paper, the contributions to the subject of pylorospasm have become plentiful and illuminating.

Symptoms.—The symptomatology of pylorospasm closely resembles that of hypertrophic stenosis; we have the same onset after two or three weeks, the same violent rejection of some but usually not all food, more or less constipation, and sometimes, but not so often, hunger stools and a variable degree of emaciation. Even visible peristalsis and a palpable tumor have been noted by some observers, from Koplik³ onward, though the tumor seems to be rather smaller. It will be seen that the only noteworthy distinction is that in pylorospasm the symptoms are apt to be less severe, because the obstruction is only partial or intermittent.

In the presence of severe symptoms the differential diagnosis between hypertrophic stenosis and spasm is almost impossible; this leads to the suspicion that intermediate conditions may exist. Holt⁵ is convinced that spasm is always present in marked stenosis, thus accounting for the sudden onset of the symptoms, as well as the diagnostic difficulties; he regards persistent spasm as indicative of organic obstruction, and doubts if it is ever purely functional. Ibrahim's first conjecture may be correct after all and the two affections may be not distantly related; this opinion of course, for the present, remains a mere hypothesis.

Certain accessory phenomena are quite constant in pylorospasm. In the first place there is an abundance of highly acid gastric secretion, and the spasm increases as the meals are made more ample. This appears to be one of the reasons why the disease does not show itself in the first two weeks after birth. We know that the supply of breast-milk is at first rather scanty, as is the secretion of gastric juice at that period; in artificial feeding we purposely give little food during the first fortnight, so that conditions in this respect are similar.

Diagnosis.—The diagnosis of pyloric obstruction, without specifying the cause, is easy enough when all the signs are present, but may be quite difficult when vomiting is the only symptom. Persistent vomiting in spite of careful regulation of the diet is very characteristic; in other digestive disturbances, vomiting is less obstinate and especially less violent.

responding, at least to a certain extent, to carefully regulated feeding. Furthermore, severe vomiting from digestive derangement is always associated with more or less diarrhea, with evidence of indigestion in the feces. Vomiting, due to obstruction farther down, in the intestine, is not always easy to differentiate, but is likely to be characterized by the bringing up of bile and intestinal contents of an alkaline reaction, which is manifestly impossible if the pylorus is occluded. Finally, the duodenal tube is very helpful when we have mastered its technique; if it passes the lower gastric orifice readily we have of course no severe pyloric obstruction to deal with.

As to the diagnosis between hypertrophic stenosis and pylorospasm, the severe cases of the latter behave so much like the former that the test of treatment is often our only resource. Even this test is probably untrustworthy, for it is extremely likely that typical stenosis has been cured by purely medical treatment (Holt⁵). Miller and Wilcox give a low acidity of the stomach contents and abundant secretion of mucin as characteristics of the former; the acidity test is certainly unreliable, as we may have a highly acid vomitus with hypertrophy; the secretion of mucin, in this condition, may also be quite moderate, for, at least in the case I2 reported, the mucous membrane was absolutely normal, without any sign of congestion, so that its secretion could hardly have been affected. Koplik³ thinks that very intense vomiting and a palpable tumor should incline us to diagnosticate hypertrophy, but his own experiences show

that these tests are not infallible. It is just as well to admit that in many cases the two affections are clinically undistinguishable.

Treatment.—As to treatment, let us begin with that of pylorospasm. Heubner's procedure is probably the best, and is generally followed, though often with certain modifications. He urges feeding at four-hour intervals, giving less than the normal number of ounces in bottle-feeding, and materially shortening the time of nursing in breast-feeding. Since Feer¹ has shown that the infant obtains most of its food in the first few minutes after being put to the breast, I limit the time of nursing to three minutes. Heubner² as well as Miller and Wilcox urge us to increase the amount of food very cautiously, even after the vomiting has been apparently brought under control. Morse⁸ differs from these authors in recommending frequent small meals; he agrees with them in giving low fat percentages when artificial feeding is necessary. Nearly all authors recommend the use of alkalies; they must be given in moderation, as they tend to retard the gastric digestion if used to excess; the alkalies do not only neutralize hydrochloric acid, but, in a measure, counterbalance this action by favoring its secretion, so that their net utility is somewhat doubtful. They are not needed in breast-feeding, and in bottle-feeding I prefer to comminute the casein coagula by using barley water. Hess4 makes the useful suggestion to feed obstinate cases through the duodenal tube; of course, this procedure is specially difficult in these very cases, but the idea has been followed

up and is theoretically perfectly sound; the mere passage of the tube is of a certain curative value.

Ruhräh² notes the three following essentials in the treatment of pylorospasm: (1) feeding with breastmilk; (2) the administration of atropine in doses of 0.03 mg.; (3) in certain cases, the employment of codeine, beginning with 0.1 mg. and increasing this, when tolerated. We may note that Holt⁵ is skeptical as to the value of any drugs in this affection.

I have found, with Ibrahim,4 that the four-hour interval is rather long, considering the very small meals which we are obliged to give; he feeds every hour or two, and I employ the latter interval myself, especially with breast-feeding; with artificial feeding, we must be more cautious, as it is absolutely necessary for the stomach to be empty between meals; low fat percentages aid us greatly toward this end, so that we can feed small meals to these babies, at any rate every three hours.

All authorities agree that the infant must be kept very quiet after meals; warm compresses, or a very lightly filled hot-water bag, applied to the epigastrium, tend to check vomiting. Lavage is often recommended, but seems irrational; it appears to me that we are chiefly interested in keeping the food in the stomach, which is only too ready too empty itself.

The above treatment is also in order, if true hypertrophy is suspected; we should give it at least a week or two of trial, for some apparently unquestionable cases of hypertrophic obstruction have been relieved in this way; we have seen that the diagnosis is often very uncertain, so that the treatment with special feeding should always be tried before resorting to more radical measures. Our only other recourse is a severe operation, which has a high mortality at this age, especially as the infant usually comes to the operating table in very bad condition. The earnest solicitations of the surgeons should not induce us to be hasty in advising operation, for not so very rarely the autopsy has shown that there was no pyloric hypertrophy present, but that the severe symptoms were due solely to spasm.

Megacolon.—Megacolon, hypertrophic dilatation of the colon, or Hirschsprung's disease, as it is variously called, was first described by the author just named.² The second name gives a good description of the anatomical findings. The disease—or malformation—has many points of analogy to pyloric stenosis, though the symptoms are naturally very different. The lesion is either congenital, or acquired very soon after birth.

Symptoms.—Meteorism and intense constipation are early symptoms, but meconium or decomposed feces do finally pass, so that a total obstruction in the intestine may be excluded. Vomiting is sometimes a prominent symptom, in other cases absent, as is usual with colonic coprostasis. The disease runs a very varying course, according to its severity; the worst cases die in the first or second year, from enterogenous toxemia or perforating ulcer of the colon, the milder ones may attain adult life. The most characteristic feature is the presence of large and externally visible coils of intestine, without anything to indicate

actual obstruction; next comes obstinate constipation, alternating with the evacuation of enormous masses of highly fetid fecal matter.

Etiology.—The authorities are not agreed on the etiology of megacolon. Hirschsprung² regarded it as a malformation, whereas Marfan¹ considers it a true partial obstruction, due to kinking of the sigmoid flexure; Ibrahim⁵ thinks that either cause may operate in different cases, but that Marfan's explanation probably applies to the majority.

Treatment.—There is no dietetic treatment for megacolon. The milder cases may be treated by palliative methods, among which enemas are occasionally useful; in the severe forms the only possible treatment consists in resection of the colon, which has rarely been successful, and is apt to prove directly fatal.

Other Spasms.—In a recent publication, Hess⁵ dwells at considerable length on pharyngospasm and cardiospasm in infants, which he sometimes finds associated with pylorospasm, sometimes with general spasmophilia. These spasms in the upper digestive tract are not very common, and the existence of true pharyngospasm is in need of further demonstration. The cardiac orifice of the stomach is, as already observed, more relaxed in infancy than in later life, as is shown by the great ease with which the stomach-tube is passed at an early age, and by the facility with which vomiting takes place. This condition is partly in relation to the normally low acidity of the gastric contents in infancy; we know that a high total acidity

and a high percentage of free hydrochloric acid are important factors in keeping the orifices of the stomach tightly closed. A more important reason, however, is afforded by the relatively feeble development of the muscular coat of the baby's digestive canal.

Singultus.—The mechanism of singultus or hiccough is not perfectly clear; it consists of a spasm of the diaphragm, caused by a reflex through the phrenic nerve, which may be started by the most varied sources of irritation. Free hydrochloric acid in the stomach seems to be one cause; mere overloading of the stomach is often a factor; in many cases it is due to peritoneal irritation, in others no exciting cause can be elicited; Hess⁵ says that it is often associated with pylorospasm.

Treatment.—The treatment of cases, due to faulty feeding, consists in removing the cause; by giving low fat percentages, we diminish the tendency to hyperacidity and spasm, and by giving smaller and less frequent meals, we avoid the factor of overloading.

Rumination.—At first sight we might suppose rumination to be a phase of vomiting, but it actually is more closely allied to singultus. We can easily understand that hiccough, in an infant, will bring up a portion of the stomach contents, which may thereupon be again swallowed. Here, too, a connection with pylorospasm seems possible or even probable, but Lust³ is not satisfied with this explanation, and thinks that rumination is more apt to be the expression of a general neurosis, as it is known to be in later life. This point of view, though by no means well established,

is yet of interest, as linking this condition with the following class of digestive disorders. Brüning observed rumination in cases of rickets and spasmophilia, and obtained a cure in connection or coincidence with antirachitic treatment.

Digestive Neuroses.—The literature on the digestive neuroses of infancy is scanty, partly because the study of the nerve functions, at this period of life, is beset with special difficulties; furthermore, recent observers have become cautious, since in the past an excessive eagerness to assume a neurotic basis for serious organic disturbances has led to great errors in theory and practice. Of this tendency I need give but one instance. by referring to the role that teething was formerly believed to play in causing the most various and serious disorders; the nervous irritation produced by the physiological act of dentition was held responsible for a long list of lesions and symptoms, which we have learned to account for on an entirely different basis, in no relation whatever to the dental apparatus. Very few physicians of standing will today attribute the most serious digestive derangements, attended with vomiting, diarrhea, and fever, to the eruption of the teeth; yet a previous generation was convinced that the occurrence at the same period of the disturbances due to weaning and the process of dentition · was not a mere coincidence, but that the former were largely due to the latter.

More scientific feeding methods have enabled us to prevent digestive disorders during the second half of the first year, so that the role of teething, as an etiological factor in organic disease, has been pretty well eliminated; on the other hand the nervous reaction caused thereby is not always trifling, especially in infants belonging to neurotic families; the fretfulness, irritability, and restlessness of many teething infants whose alimentary tract is functionating quite normally is sometimes so obvious that it cannot be dismissed as an illusion.

Some years ago Northrup reported a series of interesting cases, in infants of the age of four or five months, in whom constant exposure to nervous excitement through noise, jolting, playing, and showing off to visitors had caused rejection of food, gradual atrophy, loss of sleep, and the development of a neurotic habitus that was unmistakable; a prompt cure followed the adoption of a strictly restful mode of life. Such cases cannot be very rare, and it is astonishing that many more have not been reported. I am convinced that much of the vomiting and regurgitation that we see in otherwise healthy infants is caused by foolish habits of rocking and shaking. The rocking cradle has not yet been consigned to its merited banishment from all nurseries, and the vigor with which some mothers shake their babies up and down, especially after nursing, with the idea of keeping them quiet (!), is worthy of a better cause. In many cases these unfortunate infants simply suffer from what would be called seasickness in adults, in others the food is actually jolted out of the stomach. Many of these babies can be cured by merely laying them down to sleep, after meals, instead of harassing their nervous systems with rocking and shaking, the education of the mother proving the salvation of the child.

McClure reports an interesting case of persistent vomiting of artificial milk-mixtures in an evidently neurotic infant which was promptly checked by giving semisolid food. There were no signs of pyloric spasm or obstruction, and the stools were normal; we cannot emphasize these two points too distinctly, because they give the chief clue to the diagnosis in cases of this class.

Orchard reports a case of neurotic anorexia, in an infant of eleven months, in which treatment by starvation was successful, after gavage had failed. In the second year, similar cases are not so very rare.

CHAPTER IX.

DISORDERS OF DIGESTION (CONTINUED).

ALIMENTARY DISORDERS.

We designate as alimentary disorders those digestive disturbances that are due to the ingestion of unsuitable food; as regards infantile conditions, this practically always signifies the indigestion of cow's milk, either plain or modified. Before beginning a general discussion of this subject it will be necessary, at the risk of repeating much that has been already told, to take up, one by one, the various constituents of cow's milk and see how they may severally, or in combination, lead to derangement of the infant's digestion.

The Proteins.—The proteins rarely, if ever, give rise to trouble. We have seen that the old belief in the indigestibility of casein has been effectually shattered and that the subsequent endeavor to implicate the whey proteins has met with a similar fate. Even sick babies have very little difficulty with the proteins, for although Rubner and Heubner seem to observe impairment of the protein metabolism in atrophic conditions, Fife and Veeder find it, if anything, above normal. Holt and Levene show that

the proteins never cause disturbance, unless given in a ratio exceeding 6 per cent., and then only if the total food per day measures less than 6 ounces. Now the proteins are never given in any such percentage, and the total quantity just mentioned involves a degree of water starvation that alone is sufficient to throw the body metabolism out of gear. Under these conditions it is impossible to say how much if any of the trouble is directly due to the proteins; it seems as if they might safely be left out of consideration so far as alimentary disorders are concerned. Morse¹⁰ is one of the few leading authorities who still incline to the older views.

The Fats.—Far otherwise is the situation with regard to the fats. We have already seen that the cow-milk fats present great and, to a certain extent, insuperable obstacles to the absorptive powers of the infant even in health; we have also seen that these difficulties increase in a direct ratio to the fat percentage. From the former assumption of the entire safety of a fat proportion of 4 per cent., the same as in human milk, we have been obliged to recede; such high authorities as Holt² place the danger line very much lower in early infancy, and the foreign observers, who now call for mixtures rich in fat, content themselves with about 3 per cent.

There can be no objection to repeating a few facts, for example, Freund's¹ observation that the stools of bottle-fed infants contain about 50 per cent. of soaps—some authors recording as high as 83 per cent.—and again calling attention to the concurrent waste

of calcium and phosphorus. To this may be added a note by Meyer⁴ to the effect that these losses are increased in the presence of gastro-intestinal disease; even if Porter thinks that bad fat-absorption is due to bacterial infection of the small intestine, extending to the pancreas, this merely varies the view as to the primary etiology, but does not affect the clinical picture of fat-indigestion. We shall see that disturbance of function and bacterial infection are inextricably interwoven in the severer types of alimentary disorders.

The Carbohydrates.—The true sugars as well as dextrin play a lesser part than the fats in the alimentary disorders of infancy. We have seen that under strictly normal conditions they give rise to no trouble whatever unless given in great excess; in the percentages usually prescribed these substances are well absorbed and assimilated. The situation becomes somewhat different when the infant has become ill in consequence of fatindigestion. Finkelstein⁴ has shown that in the presence of severe intestinal lesions the power of absorbing sugar is also impaired, and that in the severest types of gastro-enteritis, sugar-intoxication predominates over the difficulties of fat-absorption and constitutes the chief danger to the infant. This matter will be gone into more fully later on; at this point it will suffice to remind the reader that the sugars, so easily assimilable by normal infants, may act as poisons under certain pathological conditions.

As regards the starches the situation is very different. The diastatic ferments of the infant are able to dextrinize only a moderate amount of starch, less than 1 ounce per day at one month of age, and not above 3 ounces at three months (Kerley and Campbell): we may regard these figures as maxima, the limit for most infants being decidedly lower. Excessive starchfeeding almost inevitably leads to the condition of starch-atrophy (Mehlnährschaden), so well described by Czerny and Keller. Of this clinical picture there is no better outline than the following after Keller.4 The infant presents a peculiarly dry skin, easily raised in folds from the muscles, which are notably rigid (hypertonic); the baby hardly moves its limbs and the heart action is feeble; there is marked anorexia, but no great disturbance of the bowels, particularly no diarrhea. The infant's color is not poor in the earlier stages, so that its appearance is no guide to the serious condition actually present; this feature, along with the lack of intestinal symptoms, is specially misleading. In the last stage there may be distinct signs of acetonemia which finally proves fatal, unless life is terminated before this by some intercurrent infection. to which these infants are extremely liable.

On careful and critical analysis it is not very clear how much of this form of atrophy is due to starchfeeding and how much to protein- and fat-starvation; probably both factors are engaged in its production, as is strongly suggested by a study of the etiology. The trouble usually begins in about the following way: the baby, which has been fed on some milkmixture, gets an attack of diarrhea; to check this the doctor or some kind friend recommends one of the starchy baby-foods, whereupon the evacuations rapidly become fewer and more solid. Repeated attempts to go back to modified milk always cause some looseness of the bowels, which is always stopped by returning to the baby-food. Finally the latter is given exclusively, matters going on fairly well for a time until the picture of starch-atrophy is gradually developed. Now we have seen in discussing babyfoods that they are almost fat-free; it is well known that the fats are only very partially replaceable by carbohydrates and most inadequately by so indigestible a carbohydrate as starch. The terminal acidosis in these infants is undoubtedly for the most part due to the disintegration of the body-fat, which Chittenden and Mendel give as the source of the acetone group of bodies; the disappearance of the subcutaneous fat is one of the most striking features of starchatrophy. Thus the role of fat-starvation is evident, the only uncertainty being whether it is accessory or only secondary.

The part played by protein starvation is not so certain, for the ratio of the proteins in baby-foods is not so very low; they are, however, chiefly vegetable proteins, only about three-fourths of which are absorbed, so that the available supply is certainly not quite up to the requirements. Furthermore, atrophic infants, as stated previously, seem to demand, if anything, rather more than the normal percentage of proteins, and we shall see that a recognition of this circumstance is the guiding principle in feeding with albumin-milk. In addition to these facts there is

good reason to believe that some of the acetone bodies are derived from destruction of the body proteins; this is more than probable in diabetes (Erving), and may well apply to other disturbances in carbohydrate metabolism. It is therefore not at all unlikely that starch-atrophy also involves a certain amount of protein starvation, though this is not yet absolutely proved.

The Salts.—Many disorders of infant digestion seem to be related to the excess of salts in most of the artificial feeding-mixtures; we have seen that the ratio of salts in human milk is considerably lower. The entire subject of salt-intoxication is, however, for the present in an unsettled state; it seemed quite well understood a few years ago, but since then more critical study has rendered doubtful some points that appeared definitely disposed of, so that I can only record the reports of a few of the numerous investigators without presuming to register a final verdict.

The whole matter revolves about the theory of what is called salt-fever. Schaps found that the hypodermic injection of 5 c.c. of a sterile physiological solution of sodium chlorid gave rise to fever which reached its acme in about eight hours and subsided within a day. Meyer and Rietschel corroborated this finding and reported similar results when large quantities of the solution were given by the mouth; they showed, on the other hand, that the fever could often be averted by adding 0.02 per cent. each of calcium and potassium chlorids to the salt solution. These experiments rendered it very questionable whether the action of

the salt solution was really toxic or the temporary fever was merely a passing chemical reaction of the body cells to the salt (Finkelstein⁵). Finkelstein⁵ does not regard salt-fever as a distinct condition, but considers it identical with sugar-fever, the trouble in both cases being a transient difficulty in absorption.

Nothmann³ shows that although the ingestion of a 1 per cent. solution of sodium chlorid can produce fever in a young infant, older infants are usually affected in this way only by higher percentages. Katzenellenbogen says that salt-fever depends much more on the concentration of the solution than on the quantity of salt. Salge² explains the greater susceptibility, in the earliest months, by the imperfection of the osmotic adjustment at that age; the liability to salt-fever is increased by disturbed intestinal absorption in consequence of starch-feeding or sugarintolerance. Weiland, who in general directly contradicts Schaps, shows that normal infants do not react with fever to isotonic (physiological) solutions of either sodium chlorid or sugar. Schlutz claims that the combination of salt and milk-sugar causes fever only in the presence of intestinal disease.

E. Schloss¹ submits the following theory of salt-fever, namely, that the solution of the surplus sodium chlorid in the body fluids causes a rise of temperature through the liberation of latent heat. This theory of course holds good only if hypertonic solutions, containing over 1 per cent. of sodium chlorid, are injected or ingested. Heim and John² attribute salt-fever to heat retention, which is a less accurate way of

stating the same proposition. Friberger notes that saltfever has usually been due to the hypodermic injection of sodium chlorid, whereas large doses may be given by the mouth without harm.

Reviewing the above reports, a mere fraction of the extensive literature on this subject, the reader will note a number of contradictions, which cannot fail to rouse a suspicion: (1) that some of these observations are fallacious, and (2) that salt-fever is not always a simple matter of osmosis and heatretention. Some of the alleged explanations are mere verbiage, masking ignorance of the true cause with a haze of scientific terms.

A rather recent communication by Samelson² has given some of the above theories of salt-fever a rude shock, from which they are not likely to recover. He proves that this disturbance is usually, if not always, due to toxins and bacteria in the water used to make up the solution, and that an 0.8 per cent. solution of sodium chlorid in fresh distilled water never causes salt-fever. This observation is in accord with the experiences derived from the many thousands of injections of salvarsan, where similar disturbances had been noted, when water that had not been freshly distilled was employed as a vehicle. Samelson's views have since been corroborated by Bendix and Bergmann as well as other authors.

These more recent findings narrow down the question of salt-fever, so far as it is related to infant feeding, to two points: (1) the injection or ingestion of a physiological salt solution, if properly performed

in the case of the former, and with moderate total amounts in either case, is harmless to the healthy infant; (2) in the presence of carbohydrate intolerance the case may be different, the salt probably adding to the febrile disturbance already started by the carbohydrates. This last point cannot be disregarded, for solutions of sodium chlorid are frequently given, hypodermically or per rectum, in cases of this group, and it is very likely, in view of the above discussion, that this treatment actually does harm instead of being beneficial, as is generally believed.

As to the pyrogenic action of concentrated salt solutions there is at hand sufficient evidence to establish it beyond a reasonable doubt. Clinically, however, this is not very important, as hypertonic solutions have not been used therapeutically and have played but a minor role in the entire discussion.

It is probable that the last word on the subject of salt-fever, remains to be written; our present know-ledge is certainly fragmentary, and the latest researches have largely impaired our confidence in what remains of a once flourishing theory.

Apart from the question of salt-fever, the salts are important factors in infant pathology because of their intimate relation to the body's water metabolism. This is due to the efforts of the organism to keep its fluids isotonic, either by retaining water, or eliminating the salts whenever the latter are present in excess; similarly a deficiency in the salt supply will result in a marked loss of water, but this condition is far less frequent. It will be readily understood that the normal

moisture of the tissues may be reduced in two entirely different ways: (1) The introduction of abnormally large quantities of salts into the body may draw off great amounts of water from the tissues; this is accomplished through the kidneys and intestine, to a less extent through the skin, and is the basis on which we administer various salts for diuretic and purgative purposes, when we desire to reduce the amount of fluid in the body; (2) the body tissues may become more or less desiccated by salt-starvation, and this may be one of the factors that cause the clinical picture of starch-atrophy from the use of baby-foods.

Under certain circumstances the body is unable to get rid of an excess of salts; this occurs, in its most typical form, when the renal functions are suspended, but may also be a consequence of certain toxic and hitherto unexplained conditions. As the salts accumulate in the body they attract sufficient water to maintain themselves, as nearly as possible, in an isotonic solution which gradually fills the subcutaneous spaces and serous cavities, producing what we call edema and anasarca. Krasnogorski gives the following three stages of this process as it occurs in infancy:

- 1. The weight becomes stationary or fluctuates: this condition is aggravated by increasing the carbohydrates or salts in the food; the normal relation of the salts and water of the tissues is evidently disturbed.
- 2. There is a large gain in weight for some days if carbohydrates and salts are given in excess; if these substances are withdrawn the weight again falls. The

tissues no longer resist the process of water-logging, and we have the condition of latent edema.

3. Clinical edema: the water is not assimilated and the body weight depends largely on the degree of edema. Meanwhile the kidneys may be perfectly normal.

All three stages are attributed by Krasnogorski to errors in the salt metabolism, and he designates them accordingly as *Salznührschaden*, *i. e.*, saltatrophy.

We shall recur to these matters when we discuss therapeutic measures, but at this point we must bear in mind one circumstance which has probably already impressed the reader: It is to the effect that the carbohydrates and salts must, to a certain extent, be added together in studying the body's water metabolism; both enter the tissues in the same way and both require definite amounts of water for their solution. In considering the disturbances of the fluid balance of the organism as a whole, the sugars and salts cannot be separated, as will be illustrated more fully later.

Let us now summarize briefly what has gone before. We have seen that the infant's digestion may undergo various derangements through the endeavor to raise it on an unsuitable food, namely, cow's milk, given plain or modified; we have also seen that the different ingredients of cow's milk are not hurtful in the same degree, the fats being evidently the usual and primary source of trouble, the carbohydrates playing a secondary part, the salts being in a subsidiary or

supplemental role, and the proteins rarely, if ever, creating disturbance, even under adverse conditions. What we next need is to combine these facts, so plainly correlated, and to construct from them a single clinical picture; to have done this successfully is the signal merit of Finkelstein and his school; however minor details may be disputed, the following framework, built up by these investigators, is likely to be of considerable permanence.

FINKELSTEIN'S THEORY.

Finkelstein⁶ takes up the general idea of Milchnährschaden, i. e., milk-atrophy, at the point where Czerny and Keller left it, and carefully analyzes its stages of development. These stages he characterizes by their leading clinical symptoms, as well as their digestive pathology. We thus obtain a steady gradation from the milder and simpler types of digestive disorder to those of greater severity and intricacy. Finkelstein also notes the various modes of treatment. as adapted to these different stages, a refinement that has been of inestimable aid in therapeutics, and, finally, he has given us a scheme of diet for the grave disturbances, which is not only founded on sound principles, but has been the means of saving thousands of infants from a condition apparently hopeless, as well as expediting the recovery of such cases as are in a less desperate state.

I. Disturbed Equilibrium.—The first symptom of disturbed alimentary equilibrium is an irregularity in the

weight curve; the baby oceasionally fails to score its regular weekly gain or alternates between good and bad weeks, with the nct result of gradually falling below normal, although the food-supply is adequate. Increase of the food, particularly of the fats, with a view to increasing the weight, always has the opposite effect, making the disturbance more pronounced.

Sometimes the stools are approximately normal, more usually they partake of the character of typical soap stools, being dry, light-colored, and somewhat constipated. Indeed, this stage is characterized by impaired absorption of the fats alone; the earbohydrates are still tolerated, if not given in excess or in the form of starch, but errors in this direction, namely, the endeavor to compensate the fat deficit with a large allowance of carbohydrates, is one of the main factors in leading the infant on to the second stage. In this respect the starches are most dangerous, whereas the malt preparations involve relatively little risk.

The temperature is no longer absolutely normal. Whereas it should range between 98° and 99.4° F., with daily fluctuations not exceeding 1°, in disturbed equilibrium we often see slight rises to 100° and morning remissions below 98°.

Treatment.—The best treatment of this stage of alimentary disturbance is, of course, a change to breast-feeding, which is sure to cure the condition promptly unless the baby is suffering from some constitutional defect like the exudative diathesis, but even in this event an improvement may confidently

be expected. Our next best recourse is to cut down the fats. If the trouble is due to venturesome attempts with top-milk feeding the fat allowance may be radically reduced by using mixtures made with whole milk. If, on the other hand, we have already been giving the latter, which are by no means a sure preventive of this disturbance, a proper adjustment becomes difficult, as we cannot cut out the fats indefinitely, skim milk or buttermilk being a very insufficient food, adapted only to very temporary use. Probably the best procedure in these cases is to go over at once to malt-feeding, letting the baby's entire carbohydrate allowance consist of dextrin and maltose. This change is usually very effective in clearing up the situation, especially if resorted to at the very commencement of trouble; the absorption of fat improves, and the stools become less typically soapy, though it must be understood that the elimination of all of the excess of calcium soaps is an impossibility with artificial feeding.

Should malt-feeding fail we have no refuge save in the temporary use of skim milk and buttermilk. In giving these we must be careful to avoid excessive carbohydrate percentages, for the reasons stated above, 7 to 8 per cent. being the limit of safety. One of the worst mistakes imaginable is the substitution of a starchy food of the Nestlé type, for the change from a condition of disturbed equilibrium to what we have learned to recognize as starch-atrophy can hardly be said to afford any advantage.

II. Dyspepsia.—The second, or dyspeptic stage, is always preceded by the one just described, but the

transition is often so rapid that, in default of a good clinical history, the dyspepsia may seem primary. Disturbed equilibrium sometimes appears to be merely a transition; this is apt to be the case when mistaken attempts are made to relieve it by increasing either the fats or carbohydrates; Finkelstein distinguishes such types as fat-, sugar-, and starch-dyspepsias.

In dyspepsia the infant is no longer able to digest the normal quantity of food, and the weight becomes stationary, or even enters on a gradual decline, occasionally interrupted by trivial gains. The temperature becomes still more irregular, with occasional rises to above 100° F.

In this stage intestinal fermentation sets in, which some attribute to exogenous bacterial infection, but Finkelstein believes to be usually due to inability of the intestine to maintain its normal flora; the fermentative bacteria, whose numbers are kept at a minimum under normal conditions, now begin to get the upper hand. When this takes place a catarrhal condition of the intestinal mucous membrane follows, as a matter of course; the stools may still be only moderately numerous, but showing every evidence of greatly impaired fat digestion, or may become truly diarrheal.

Though fat-dyspepsia sounds the key-note during this stage, there are already some signs of impaired absorption of the carbohydrates; the fermentative processes attack the ingested sugars, leading to a considerable formation of gases, with resulting colic; it is very clear that the readily fermentable starches, under these conditions, will give more trouble than milk-sugar and cane-sugar, and these again more than malt, which does not ferment easily.

The symptomatic dividing line between dyspepsia and the next stage is not always sharply defined; the distinction is often rather in degree than in kind; but the very slight and gradual loss of weight in merely dyspeptic disturbance is a valuable criterion.

Treatment.—Breast-feeding is here also the sovereign remedy, sure to cause rapid improvement. If there is no opportunity to turn to breast-feeding we must resort to skim milk or, still better, buttermilk, but without the addition of carbohydrates, for fear of hurrying the disorder onward to the next stage. Superior to either is the substitution of malt for the ordinary sugars; it is in this condition that Keller's malt-soup has proved so eminently successful; we have shown how effective dextrin-maltose mixtures are in checking the progress of intestinal fermentation. Keller's method is of course, as previously mentioned, merely a temporary device, to be employed for some weeks only.

If the dyspeptic stage shows no improvement within a week, the infant is in imminent danger of going over into the following condition.

III. Atrophy (Decomposition, of Finkelstein).—In various parts of this manual I have employed the word atrophy to translate, rather inadequately, the German term Nührschaden, which cannot be rendered by an exact English equivalent. The third stage of impaired nutrition in the classification of Finkelstein, called by him Decomposition, is recognized by him as

identical with what has always been called atrophy. In this stage anything like proper nutrition has become impossible; the loss of weight is rapid, an ounce or two per day; the infant cries incessantly, from hunger as well as general discomfort, and manifestly suffers from starvation, as it takes proffered food greedily. The skin is pale, the oral mucous membrane reddened. The temperature becomes very irregular, with a marked tendency to be subnormal, going down to 97° F., and even lower.

The evacuations vary greatly; they may not depart so very much from the normal, but this is rare; more commonly they are soapy, or evidently undigested; fat-diarrhea and tarry (bloody) stools are not uncommon. The pulse is rather slower than normal; the respiration at first shows prolonged expiration, then irregularity, occasionally being of the Cheyne-Stokes type. There is no evidence of any renal lesion, but an edema or anasarca of metabolic origin may nevertheless be present. Sometimes the infant becomes cyanotic.

This stage often proves fatal, either from sudden collapse or from acute inanition, with a rapid fall in the weight and temperature that lasts for a few days, or from an intercurrent infection, to which atrophic babies are particularly liable. Finkelstein differs from some authors, who think that an infection is essential to a fatal outcome; he regards the mere atrophy as a sufficient cause of death.

In the stage of atrophy the absorption of fat is reduced almost to zero and the tolerance to carbohydrates has begun to suffer severely; concurrently there is more or less disturbance of the salt-metabolism, as is shown by the tendency to edema and anasarca. It is not improbable that the worst cases may exhibit a diminished tolerance for proteins, though this has not yet been proved.

Treatment.—Even the resort to breast-milk may fail to save a severely atrophic infant; indeed, the end may be hastened by full breast-feeding, because even the human milk-fats are now badly tolerated. We must often begin with small allowances, drawn with the breast-pump, not giving more than an ounce or two at a feeding to commence with, and in the worst cases even less. As to artificial feeding, the treatment outlined for dyspepsia, especially the method devised by Keller, may be successful in cases that are not too far advanced; in some of these we may eventually return to an ordinary whole milk formula. In bad cases the Keller treatment is only temporarily successful, sometimes even harmful, from the high ratio of carbohydrates, and a relapse invariably follows any attempt to return to a really sufficient diet, so that the saving of the baby becomes a problem of extreme difficulty. In the very worst cases, Keller's malt-soup utterly fails to achieve any improvement, and the infants are lost unless breast-milk is at hand to save them.

It will not be amiss to add a few words of caution against treating these cases with a preliminary course of starvation. This treatment is highly effective, if limited to twenty-four hours, in intestinal infection or intoxication, but it is utterly out of place in atrophy, of which a form of chronic starvation is the essential feature. This is clearly pointed out by Rosenstern.²

IV. Intoxication.—The relation of the stage of intoxication to the three others is very far from obvious, and represents the vulnerable portion of Finkelstein's theory. He regards it as essentially a continuation of the preceding stages, but we do better to regard it as a sequel or complication that may follow in the wake of either dyspepsia or atrophy.

In intoxication we encounter a feature that has so far been relatively inconspicuous, namely, an almost total intolerance for sugar; on the other hand the power of absorbing fat is somewhat better than in atrophy, and more like that in mere dyspepsia. The protein digestion is practically unimpaired.

Symptoms.—As to the general symptomatology the most marked feature is fever, of a very irregular type, but sometimes reaching very high degrees. The respiration is deep and hurried, often panting, so that Finkelstein feels justified in speaking of asthma dyspepticum, a symptom first described by Henoch. The pulse is rapid and feeble, so that collapse seems imminent; the blood shows a moderate leukocytosis, rarely exceeding 30,000.

Diarrhea is present, in varying intensity, ranging from moderately frequent undigested stools to very numerous rice-water evacuations; if collapse sets in the diarrhea may stop. In the urine we find albumin and casts, sometimes also sugar, but in this event always lactose or galactose, never dextrose, as in diabetes; the lactosuria furnishes evidence that the food sugar

is badly assimilated, even when absorbed, because of impairment of the glycogenic function. There is of course a rapid loss of weight, so that the infant soon becomes extremely emaciated.

Symptoms referable to the nervous system are common. Somnolence is frequent, and may pass on to stupor and coma; cataleptic conditions are often encountered, as are convulsions; the nervous symptoms are not always in proportion to the gravity of the disease. According to the predominance of one or another group of phenomena, Finkelstein divides alimentary intoxication into several clinical types, such as the choleriform, soporous, hydrocephaloid, and asthmatic.

In reviewing the above account of the stage of intoxication the American reader will have no difficulty in recognizing the clinical picture, known to him as acute enterocolitis, summer diarrhea, and cholera infantum. The centre of interest in this connection lies in the stand-point of Finkelstein, that we are dealing with a terminal stage in an alimentary disturbance, for which the stages of disturbed equilibrium, dyspepsia, and atrophy have been preparatory. It is undoubtedly true that intoxication is nearly always preceded by other types of digestive derangement, but I must agree with the many authors who do not regard it as a mere terminal outcome. One very weak point in Finkelstein's position is the circumstance that the fats are usually better tolerated in the fourth than in the third stage; this is certainly very curious and unaccountable, demanding an explanation which

Finkelstein has failed to supply. Furthermore, a whole series of symptoms points to a bacterial infection as the essential factor in causing intoxication; among these I may mention the fever, leukocytosis, cerebral phenomena, and evidences of nephritis, not to speak of the fact that a number of demonstrated bacterial diseases give us exactly the same clinical picture. I therefore regard the attitude of Talbot¹ as reasonable when he considers the stage of intoxication an exogenous infection, grafted on to a digestive disturbance, but not a merely higher degree of the latter. Finkelstein himself has admitted that infants who suffer from the earlier stages are exceedingly subject to the invasion of pathogenic microörganisms; he commits himself seriously when he speaks of the fourth stage as an auto-intoxication. Modern views are becoming very conservative on the subject of auto-intoxication; no less an authority than Adami doubts the very existence of enterogenous forms of this affection, declaring that the action of invading organisms is absolutely essential to the production of toxic symptoms in intestinal disease. Clincially, however, Finkelstein's view has some justification; he stands on fairly firm ground when he maintains that a previously disordered digestion always precedes the development of the stage of intoxication, and that bacterial infection cannot take place if the mucous membrane of the gastro-intestinal tract is in a healthy condition. We have seen that the predominance of the lactic acid bacillus, the controlling germ of the normal intestinal flora, is inimical to the growth of other microorganisms;

it is therefore altogether likely that a severe disturbance of function, and a catarrhal condition of the mucous membrane, are indispensable prerequisites for an alteration of bacterial conditions and the development of the processes here under discussion.

Whatever conclusion may eventually be reached, one important observation of Finkelstein is likely to hold good, namely, that the carbohydrates act as the toxicogenic substance in alimentary intoxication and that their withdrawal is the chief requirement of successful treatment. This is in itself no inconsiderable achievement, and in support thereof, Finkelstein and his followers have furnished ample proofs. The studies on salt-fever, previously discussed, although they have not hitherto led to satisfactory conclusions, do not in any way invalidate the main proposition, but in fact strengthen it, since it has generally been accepted that the carbohydrates and salts must be added together in considering the disturbances of the water metabolism, which play so important a role in alimentary intoxication as sometimes to come into the foreground, as in the choleraic type.

Treatment.—The treatment of this condition is a logical sequence of the etiological determinations outlined above, but will not be detailed in this place, because we desire to avoid repetition. We can take it up to better advantage when we proceed to discuss the various forms of intestinal infection in the following chapter, where the clinical picture, which has just been sketched, will be presented more fully from the bacteriological point of view.

Finkelstein's researches have led to far-reaching and epoch-making progress in various other directions. They have administered the quietus to the view that casein causes digestive trouble, and have led to the intensive study of salt-fever, discussed in a previous paragraph. The best result has been a more rational and successful treatment of the most severe forms of gastro-intestinal disease, worked out through clinical experience and not based on preconceived theories. We shall see that the most recent investigations have furnished a scientific basis for the good results that have been obtained, but the net outcome is one more example of the superiority of bedside observation, when intelligently conducted, over uncontrolled laboratory research and mere reverence for authority.

CHAPTER X.

DISORDERS OF DIGESTION (CONCLUDED).

INFECTIOUS DISORDERS.

The third great group of digestive disorders is caused by the invasion of the gastro-intestinal tract by pathogenic bacteria. Practically these bacteria gain access to the body only through one source, namely, the food; therefore, from the nature of the case, the diseases here considered must be chiefly associated with artificial feeding. They are conveyed to the infant either by infected milk or through uncleanliness in feeding, whereby otherwise innocuous milk is inoculated with disease germs. This is the reason why, in a previous chapter, detailed advice was given as to scrupulous cleanliness in the manipulation of bottles, nipples, and the other paraphernalia that are employed in bringing up infants on cow's milk.

Through bacteriological researches it has now been definitely ascertained that there are several forms of infectious intestinal disturbance in infants; if we here decide to discuss them together it is because their differentiation is difficult, their symptomatology is similar, and their treatment not very diverse, whereas the prophylaxis is practically the same for all.

Etiology.—Let us begin with a consideration of the predisposing causes. First among these is the presence

of one of the milder degrees of alimentary indigestion. It was formerly believed that infectious diarrhea would attack a normal infant almost as readily as one whose digestive apparatus was already more or less disordered, but experience and investigation have shown that this is, strictly speaking, not the case. For practical purposes, however, this consideration is not so important, as it would seem; undoubtedly the majority of bottle-fed babies are not raised according to the most approved methods, and a very large percentage is always suffering from the condition described as disturbed equilibrium. We have seen how this is apt to pass on, in some cases quite rapidly, to the stages of dyspepsia and atrophy, which admittedly predispose to bacterial infection. It is therefore not remarkable that even badly managed breast-fed babies rarely suffer from this disease, since the chief predisposing factor is usually lacking in them, even though their intestinal functions may not be quite normal.

The second predisposing cause is warm weather, which does not by any means imply intense or tropical heat, but does require a certain degree of atmospheric humidity to become effective. The sensible temperature, a mathematical function of temperature and relative humidity, is more important than the ordinary temperature as indicated by the dry-bulb thermometer. Dry heat is comparatively harmless. Thus a temperature of 80° with 80 per cent. of humidity represents the same sensible temperature as 95° with 40 per cent. of humidity, and is equally injurious.

So influential is this factor that infectious intes-

tinal disease is generally spoken of as summer diarrhea, though similar cases occur, but far less frequently, in the cooler months. In the days when infant hygiene was indeed in its swaddling clothes, this country, especially the larger American cities, had a most unenviable reputation for a high infant mortality from diarrheal diseases during the summer season. This was attributed in part to American heedlessness in general, but in part most justly to the heat and humidity, which we had, in those days, no means of guarding against. As a matter of fact, the summer of our three leading cities is fully 8° F. warmer than that of the three greatest European capitals, and the humidity is, if anything, also slightly higher in the former.

Among the first to present reliable data on this subject was Seibert, who constructed a chart showing the death rate from diarrheal diseases in infants and young children, in New York City, month by month. The rate remains uniformly low until the middle of May, when the mean temperature has reached 60° F., a steady rise then takes place until the latter part of July, when the average temperature is highest; then a decline begins, which grows rapid in September; in early October the mean temperature has again fallen to 60°, but this month still shows a higher death rate than the months from November to April, because of a number of chronic cases that hold over from the summer to succumb in the fall.

Some years ago I³ drew up a similar chart, based on the sensible temperature, the details of which do not differ materially from those of Seibert. The dangerline, according to the sensible temperature, is a little lower, about 55° F.

European cities, north of the Alps and Pyrenees, furnish similar death-rate charts, but with greater annual variations. The ordinary north European summer has a mean temperature not so very much above 60°; the mortality therefore rises more slowly and does not attain its maximum until August, because the type of infection is not so acute nor so rapidly fatal. In cool summers, which seem very chilly indeed to an American, the rise in the death rate in such cities as London, Paris, or Berlin is very small; in hot summers, as in 1911, the chart resembles that of American cities. I may note that in New York City, in recent years, not only has the crest of the mortality curve fallen considerably, but the maximum has shifted to August, even when the hottest weather occurred in July. This would indicate that the fulminant cases of cholera infantum have been largely replaced by the subacute and less rapidly fatal forms of intestinal infection, and I am sure that most local physicians will bear me out in this opinion. As our climate has not changed, some recent summers, as that of 1911, having indeed been exceptionally warm, it would appear that the evil influence of mere heat is, to a great extent, controllable, and that its role is merely predisposing, not directly provocative of diarrheal disease.

There are still many authors, such as Schereschewsky in this country, and Rietschel in Germany, who consider hot weather, and especially overheated lodgings, as a direct exciting cause; they regard the most acute forms of cholera infantum as phases of heat-stroke. Aside from the symptomatology, in which these cases do not in the least resemble heat-stroke, it has been often noted that the greatest mortality does not coincide with the hottest weather. It should be remembered that heat-stroke consists of simple hyperpyrexia, induced directly by the influence of intense heat, which the organism is unable to counteract by radiation and perspiration. The following records for New York illustrate the true relation of these matters:

Months.	July, 1911.	Aug., 1911.	July, 1912.	Aug., 1912.	July, 1913.	Aug., 1913.
Temperature, Fahrenheit.	77	73	76	72	76	73
Sensible temperature	68	67	67	65	68	66
Deaths from heat-stroke .	535	17	75	6	44	12
Deaths from diarrheal diseases (under five years)	809	1034	771	925	598	867

We see that the deaths from heat-stroke coincide with the high temperatures, but that the mortality from diarrheal diseases shows no such intimate relation. The high heat-stroke figure for July, 1911, was caused by an extraordinary hot spell during the first half of that month, but at the same time the infant mortality was about as usual.

The best disproof of the heat-stroke theory is furnished by the statistics of New York City, where the decline in morbidity and mortality from summer diarrhea has been enormous, though the climate has not changed, and the rooms of our tenements are pretty much the same malodorous sweat-boxes that they were ten or twenty years ago.

Hot weather acts chiefly as an indirect cause, in that it favors the growth of pathogenic and putrefactive bacteria in the milk. If this article of food could be obtained in a nearly aseptic way and kept cool, so as to hinder the growth of the relatively small number of contained germs, the harmful effects of mere warm weather could be almost entirely obviated.

We pass on to the one exciting cause, the bacteria in polluted milk. We have already discussed the question of milk-supply quite fully, and need only repeat that in every community the decline in infant mortality has strictly coincided with the improved supervision of that food. Results that at first seemed incredible were reported from such cities as Rochester, New York, which was one of the first to adopt a system of really adequate milk-inspection. Since then the same effect has been observed in every community where similar methods have been adopted.

Rietschel is undoubtedly right when he claims that mere dirt in the milk does not cause intestinal infection, but, after all, this objection is purely academic. All impure milk, as well as some that passes muster, is liable to contain disease germs, and this is indeed the chief argument for the enforcement of universal pasteurization. These views are gaining ground so rapidly as to convert even those authorities who, on a theoretical basis, prefer high-grade raw milk as in

many respects a better food. Many of the former rawmilk advocates are now advising pasteurization of even the best quality of milk, at least during the summer months.

Since pathogenic bacteria are the direct cause of intestinal infection, let us pass on to a study of their various species and manifestations in detail.

In 1898 Shiga found a microörganism called the Bacillus dysenteriæ in cases of ordinary dysentery, as met with in Japan, thus showing that there were two forms of so-called tropical dysentery, the bacillary and the amebic, Shiga's bacillus being the cause of the former. Soon after Flexner, in the Philippine Islands, found a somewhat different bacillus in the same disease; this variety of the Bacillus dysenteriæ is distinguished as the type Flexner, or Flexner-Harris. Since then another variety, called Type Y, has been found, and Ohno, reporting from the Philippines, shows that there are at least fifteen varieties of this bacillus, all closely related to one another in morphology and pathogenicity.

A few years afterward the above discoveries were brought into relation with the subject before us. In 1902 Duval and Bassett found the Bacillus dysenteriæ in the evacuations of cases of summer diarrhea in infants, a discovery that soon received ample confirmation. A comprehensive study of this question is furnished in the report of Flexner and Holt, where cases due to all three of the above-mentioned types are recorded, the culture findings being confirmed by serological tests. A few special points may be

emphasized. In the first place, dysentery is reported in breast-fed infants: in these cases the infection cannot have taken place through the milk, but must have been transmitted through uncleanliness in some other respect. Some cases were observed in the cool months, and ran the same course as the typical summer infections. A previous lesion of the intestinal mucous membrane seems to be essential; in the normal intestine the bacilli of dysentery lead a merely saprophytic existence, ready to create a severe disturbance as soon as a digestive disorder has set in; this is in accord with the findings of Finkelstein, reported a few years later. It is doubtful if the bacillus can be isolated from the evacuations of normal infants except such as have but recently recovered from a diarrheal attack; the bacillus is none too numerous in the acute cases. and its cultivation depends greatly on the skill of the investigators, the most expert of whom have found it in over 90 per cent. of all cases.

In temperate elimates the Flexner type is commonest, such is also the testimony of Weaver and Tunnicliff and Wollstein; many German observers have not found the Shiga type at all (Knöpfelmacher, Leiner). Of interest is the recent finding, in many cases, of a gas-forming bacillus; in the present series of researches this bacillus was purposely eliminated, as interfering with the cultivation of the Bacillus dysenteriæ, and thus a matter of great importance was ignored by the earlier investigators.

Some years afterward Kendall and Smith came out with an interesting report in which they claimed a

pathogenicity, similar to that affirmed of the dysentery bacillus, for the gas-forming bacillus (Bacillus perfringens). This germ is sometimes found in immense numbers in diarrheal stools, and may be easily isolated by cultivating in milk that has been heated to 80° C. (176° F.). The Boston observers have been dwelling on this point ever since, and confirmation of their findings would be very desirable, as in some epidemics the dysentery bacillus is either uncommon or very hard to isolate. Among other bacteria, streptococci have been charged with producing summer diarrhea (Knox and Schorer), but their role, as well as that attributed to the ubiquitous Bacterium coli, cannot, for the present, be absolutely determined.

Kendall has shown that different bacteria prevail in different years, but R. M. Smith points out the difficulties in the way of the practical utilization of this circumstance, since the symptoms show no corresponding variation.

Pathology.—According to the various development of the intestinal lesions, Flexner and Holt distinguish four types of pathological findings:

- 1. The lower ileum and cecum are lined with an extensive pseudomembrane, consisting of necrotic tissue, cells, and bacteria, but little or no fibrin (typical dysentery).
- 2. There is extensive hyperplasia of the lymphatic tissue, but little involvement of the mucosa and a few small ulcers.
- 3. There is superficial necrosis of the whole mucosa, but no pseudomembrane.

4. The most frequent type: there is only slight hyperplasia of the lymphatic tissue. This is the common finding in cases that have been already ill before the acute symptoms appear, and which succumb rapidly.

Rotch³ claims that the bacillus of dysentery is specially associated with ileocolitis and is not likely to occur in merely fermentative diarrhea; he asks us to distinguish between these pathological types as being etiologically different. In this he disagrees entirely with the findings of Flexner and Holt, whose position is being more and more sustained as observations multiply. Any sharply drawn lines, among the four types given above, are quite artificial and clinically useless; the old collective term of summer diarrhea, without histological specifications, is still a practical and adequate designation.

Symptoms.—We have already studied the symptomatology of the severe cases in describing alimentary intoxication in the preceding chapter, and have noted that there is no clinical difference between that disorder and the one we are now discussing. It will, however, be of advantage to go into some of the details of the specific bacterial infections. Michael observes no difference in symptoms between the Shiga and Flexner types of infection, and La Fétra and Howland agree with him; Park¹ differs from them in considering the Shiga type more severe. Knox goes into the diarrheal symptoms more minutely and finds that mucus is always abundant if severe intestinal lesions are present, and that the amount of

blood and pus in the evacuations is fairly proportional to the extent of ulceration; small quantities of pus may, however, escape notice. Blood and pus rarely occur separately, and are met with only after the colitis has lasted two or three weeks; we have seen that the severest cases do not last so long, proving fatal before actual ulcers have time to form. Nephritis is common in the worst types, and edema is an ominous sign that is independent of the degree of the nephritis, and may appear without it (Potter).

The usual clinical picture may readily be grasped from what has been said. After a period of unstable weight, with soap stools or moderate diarrhea, corresponding to what has been called disturbed equilibrium or dyspepsia, the infant suddenly goes over into the stage called either intoxication or intestinal infection, with or without a short intermediate stage of atrophy. There is more or less irregular fever, the loss of weight is very rapid and continuous, amounting to a pound in a few days; the stools are frequent, at first diarrheal, ten to twenty or even more per day, finally becoming watery (choleraic). At this stage collapse is apt to set in, with possibly subnormal temperatures, which are of unfavorable import. From this point on, three different courses are possible: (1) the infant may succumb in a few hours or a few days at the most; (2) the diarrhea may cease, the fever gradually subside, with a marked steadying of the temperature and weight curves, and the patient progress to a slow recovery; (3) the symptoms may undergo a slight abatement, but diarrhea continues, the stools gradually

begin to show quantities of mucus, later on blood and pus, the weight still continues to fall, though a little more slowly. Cases of the third group may either recover finally, after a long convalescence, during which they are in constant danger of a relapse, or they may go on to a chronic colitis, and atrophy (Decomposition of Finkelstein), with a fatal termination after some weeks or months.

Prognosis.—Most of the statistics that relate to the prognosis of infectious diarrhea are vitiated by the error of including all the cases of intestinal disease that occur during the summer. Now if we include the relatively harmless diarrheas met with in breastfed infants, as well as the mild cases, with nearly normal temperatures that represent merely intestinal dyspepsia, the mortality is only very moderate. So soon, however, as we limit our statistics to the truly infectious cases, adopting as our criterion a repeated rise of temperature above 101° F., the prognosis becomes very doubtful.

It is evident that we must subject the statistical reports to a searching analysis. Thus Dunn,² using the criterion just mentioned, classifies 111 out of his 620 cases as of the infectious type; of these 29 did not return, reducing his actual clinical material to 82 cases; of these 12 are recorded as dead or unimproved, the latter being almost equivalent to a death sentence in this disease, so that we have a probable mortality of 14.5 per cent., whereas the unanalyzed statistics for 620 cases yield a mortality of barely 2 per cent. In the report of Flexner and Holt we find an account of 207 cases in whom the bacillus of dysentery was

actually demonstrated, with a death rate of 35 per cent. The last figure fairly represents the mortality of bacillary infection under the then prevailing methods of treatment. Accurate and carefully analyzed reports, based on the more modern therapeutic procedures, are not yet extant, but the prevailing impression, as well as the picture afforded by recent urban mortality statistics, seems to indicate that during the last few summers the death-rate has been considerably reduced.

A point that appears to be of some prognostic value is mentioned by La Fétra and Howland, namely, that the previous condition of the infant is a very useful guide; there cannot of course be any doubt that a baby already suffering from atrophy has a much poorer chance than one that has been merely dyspeptic; we may therefore allow the previous history to modify our prognosis in the individual case. It goes almost without saying that the outlook also varies according to the intensity of the symptoms; exceedingly frequent watery stools, meningeal symptoms, anomalies of the respiration, and edema are all unfavorable signs, whereas the prognosis is still very hopeful if only gastro-intestinal manifestations are present. Opportunities for adequate treatment also influence the outcome more favorably than in almost any other disease.

Prophylaxis.—The prophylaxis of intestinal infection is intimately related to the question of the milk-supply, which has already been discussed. Next come the cleanly preparation of milk-mixtures and the proper care of the containers, bottles, nipples, and so forth, besides an ample provision of ice,

especially in the summer months. The suppression of the house-fly at present occupies the centre of the stage, but is of secondary importance if the baby's food be kept carefully covered; much of the fly agitation is pure sensationalism, and due to an endeavor to find a short cut to sanitation; if the premises are kept clean the flies cannot carry dirt.

Certain other measures are of great value, but require the cooperation of the community and form a part of the great social question. First come proper home surroundings, particularly for the poorer classes; this includes the whole field of tenement construction and supervision, and belongs to the State and Municipal authorities. Next comes provision for purer air than can be found in the midst of our great cities; in this regard our sea-coast towns have exceptional opportunities which have been utilized, so far, in a manner by no means equivalent. Much can be accomplished by means of seaside parks, water excursions, and recreation piers; a great deal of the progress achieved in such cities as New York and Boston can be attributed to the development of these resources, but the greater part remains to be done. Even more important is the instruction of mothers in proper methods of infantfeeding and hygiene: (1) in the inculcation of nursing, whenever this is at all possible; (2) in teaching each individual parent the correct methods of milk-modification in the home, or supplying the proper mixture, the latter an inferior plan, because too apt to be handled in routine fashion. If we succeed besides in doing away with certain feeding superstitions that

are popular with the masses and banish the dirtcollecting pacifier, still more will be gained. These general measures can only be carried out through milkstations which should, however, not be mere dispensers of milk, but should be run on the lines of well-equipped hospital dispensaries; they are indeed apt to work best if affiliated with the pediatric service of a hospital. Milk-stations that are merely presided over by a physician, or still worse a trained nurse, and leave the details of feeding, as well as the care of the home. to ignorant mothers, are of relatively little value; home visiting is an absolutely essential part of the scheme. Still, I would not discard the old-fashioned milk-stations as entirely useless, for they have at least the merit of controlling the quality of the milksupply.

Prominent among prophylactic measures, and almost self-evident, in view of the etiological factors, is the prompt and adequate treatment of infants suffering from disturbed equilibrium and dyspepsia, before their intestines become infected. This must, for the present, remain largely a vain desire. Some mothers think that a moderate diarrhea is normal, others believe that their babies are ill from teething, still others resort to starch-feeding, with the disastrous results before mentioned; last, but not least, come the parents who go for advice to solicitous relatives, or some self-declared lay expert, in fact to anybody except the doctor. I regret to add that instruction in infant-feeding in the medical schools is still not what it should be, and is very often far behind

the times; too many teachers stick to their own hobbies and do not give the student the benefit of the latest researches; in some medical schools the study of physiology is neglected in favor of the practical branches which, after all, rest on a thorough knowledge of the functions of the normal body. Thus many members of the medical profession, especially some older ones, whose active practice has not permitted them to follow recent studies, are but imperfectly equipped for this exacting field of work.

Treatment. — Before discussing medicinal and dietetic treatment let us briefly consider such general measures as are agreed on by all. First comes removal to a purer atmosphere; it is not so important that the temperature should be low as that the infant should be taken from surroundings that often are positively fetid. We have just noted some means of accomplishing this in discussing prophylaxis. of all is the floating hospital, first adopted in Boston and New York, where fresh air and clean food are added to expert medical attention; I wish to emphasize the last, as without it the excellent results achieved by these institutions would be lacking. In the same class I would place the seaside hospitals, now becoming quite numerous; they have the additional advantage that the infant need not be returned at night to the noisome tenement, but unfortunately the much greater disadvantage that they are more expensive to maintain, and of course very limited in capacity. Recreation piers, with attached milk-stations, under medical control, fill the great gap that is left open by the aforesaid facilities; they are capable of as much extension, as there is waterfront, and inexpensive of maintenance, after the somewhat heavy cost of construction has been covered. There are now ten of these recreation piers, distributed along the edge of Manhattan Island, and providing for at least as many thousands of babies.

Local treatment of the intestine is of limited value, chiefly because the affected portion is practically inaccessible. Lavage is useful if vomiting is a prominent symptom (Lucas²), but this usually ceases promptly when a suitable diet is adopted. The value of colonic irrigation has been overrated; it is of greatest use in those cases that pass on to a chronic colitis (Rotch³). Medicinal treatment has comparatively limited applicability. Many authorities initiate treatment with a purge; for this purpose calomel, in doses of $\frac{1}{2.0}$ to $\frac{1}{1.0}$ grain, frequently repeated, was once the universal drug; it is, however, no better than easter oil, and far more irritating (Abt), a point worth considering in the case of an already inflamed intestine. Castor oil may be of use in cases on the borderline, where fermentation still seems to predominate over infection; I think it unnecessary, and even contra-indicated when the evacuations are watery and excessively frequent.

In very severe cases, opium, though largely discredited in recent years, is still, in my opinion, the most valuable drug; if given in a dosage of 1 minim of paregoric to each kilogram (2 pounds) of the infant's weight, and carefully watched, it is of the

greatest possible value and cannot do harm. Alcohol given in the form of 10 to 20 drops of whisky, diluted with ten times as much water, has also fallen into unmerited disfavor; in threatening collapse it is almost indispensable. In emergency such drugs as caffein and camphor are called for; I prefer the latter as more rapid in action, and give at least \(\frac{1}{4}\) grain, i. e., 5 minims of a 5 per cent. solution in olive oil, hypodermically in fairly frequent doses. Caffein-sodium benzoate may be given in similar dosage, alternating with the camphor, until the infant has been tided over its collapse.

For the intestinal condition, bismuth has always been a popular remedy, but its results are often disappointing; as it is merely astringent, and has no effect on the bacterial growth, it is not remarkable that it should frequently fail to be of benefit. In the later stages, when colitis is the most prominent feature, bismuth is more likely to prove useful, but I have had greater success with tannin, especially if given as an albuminate, such as tannalbin.

To compensate for the great loss of body fluids, we often have to resort to saline infusions or the Murphy drip. We must take care to use a solution that is strictly isotonic (physiological), otherwise we run the risk of adding the complication of salt-fever, previously referred to.

Serotherapy has turned out to be of little use. The favorable reports of Rudnik and Auché and Campana, as well as of some American authors, date from the early experimental period. At the same time we

obtained adverse reports from La Fétra and Howland, Hastings, and Knox, which have not been refuted by subsequent experiences. There is at present no good prospect of an efficient specific therapy.

Our main reliance in the treatment of infectious diarrhea is on dietetic measures. It was long ago recognized that treatment by prolonged starvation was likely to hasten the fatal outcome, and in the days when the true etiology of summer diarrhea was still unknown many of us learned to go over to albumenwater, after a starvation period not exceeding twentyfour hours. The preparation of albumen-water has been described on page 177. If made up with barleywater it contains about 1.5 per cent. each of proteins and carbohydrates, with practically no fat. In the light of our present knowledge it is not remarkable that a certain measure of success attended this mode of treatment; we must remember that in those days egg-albumen was considered safer than the dreaded casein of cow's milk.

It has long been recognized that two dangers must be avoided in the feeding of these cases. Coolidge has contributed an article on the starvation method which sheds much light on one error in treatment; one of the worst things we can do is to give water or even barley water freely until the stools are approximately normal; under this treatment the patient is likely to do badly. The next point is noted by Potter and others, and is in accord with much old and all modern practice, namely, to start in with a fairly ample protein diet within one or two days. There is at present little disagreement on this question, but Lucas² not so long ago still recommended a rather long period of preliminary starvation, followed by a very weak formula, which he made very low in fats. We have learned that the fats need not be kept so very low, and that we may safely give one-half to two-thirds of the normal percentage, provided we keep down the carbohydrates.

Dunn³ and Clock recommend the feeding of living lactic acid bacilli in a fat-free milk, but Dunn,⁴ in a later communication, admits that this procedure has not been very effective. Dunn,³ Decherf, as well as others recommend buttermilk. Whereas buttermilk and skim milk have, to balance their defects, at least an adequate protein content, this is not the case with whey, which has also been extensively given in summer diarrhea. We know that whey is essentially a lactose solution, with minimal amounts of both proteins and fats. Leopold and Müller offer evidence to show that whey rather favors intoxication in this condition instead of relieving it; we shall see that its administration is diametrically opposed to modern views, and based on a fundamental error.

To Finkelstein⁵ belongs the credit of first observing that the carbohydrates are the food element that causes the symptoms of intoxication in the severest forms of intestinal disturbance, and that withdrawal of sugar brings about a cessation of the fever, whereas a return to that food inaugurates a renewed rise of temperature. A really good explanation of this phenomenon has hitherto been lacking, and the search for

one led to the study of salt-fever as a possible factor: we have seen that this investigation has led to little of value so far as the main proposition is concerned. I would suggest a simple way of accounting for the pyrogenic action of sugar in intestinal infection, namely, that the growth of the bacteria requires a certain supply of carbohydrates, and that a medium poor in sugar checks their development. This would explain why sugar causes no trouble in the absence of these germs, even when the digestion of the fats is greatly impaired. As to the moderate intolerance for sugar that is witnessed in milk-atrophy, there is one very considerable difference to be noted, namely, that the sugar does not appear to act as a toxic substance, and especially causes no rise in the temperature. which is indeed apt to be subnormal. The so-called intoxication-fever is best regarded as entirely due to bacterial toxins, and only to be indirectly referred to the sugar itself, insofar as it favors bacterial growth.

Though Finkelstein seems to have missed the true significance of the intoxication-fever, he nevertheless, through careful clinical observation, evolved a method of feeding, unquestionably superior to any previously known; by working along entirely new lines, he devised the modification called albumin-milk, the preparation of which has been given in detail on page 118.

Before going further, we must make note of certain precautions in feeding with albumin-milk. In the first place the milk must be prepared strictly according to specifications, particularly avoiding lumpiness (Finkelstein and Meyer²); there have been a number of recent endeavors to prepare this food, by adding commercial casein to buttermilk, but the resulting product is not Finkelstein's albumin-milk, being nearly fat-free, aside from the uncertain food value of the desiccated casein.

Some authors give albumin-milk too liberally; the limit should be six or seven ounces per kilogram of weight, or three ounces per pound, a very ample allowance, by the way, and too much for severe cases to begin with. Some add too little buttermilk; Finkelstein⁷ insists on the proportion of one-half; in the same communication he admits that the fat-ratio varies between 2 and 3 per cent. The latter figure is rather high, but may be avoided by using a rather low fat (3.5 per cent.) milk, as raw material for the curd extraction. Following this idea further a number of American pediatrists advise the preparation of albumin-milk from skim milk, instead of whole milk. thereby lowering the fats to a minimum; it is doubtful if this is ever necessary or even useful; at any rate it cannot be kept up for any length of time, for there is great danger, on so low a diet, of the continuance of the rapid loss of weight, which of itself constitutes a serious menace to the baby's life.

Treatment with albumin-milk must not be delayed until the infant is moribund, but begun as early as intoxication appears, allowing a preliminary starvation period of twenty-four hours. In severe cases we may begin with a teaspoonful every two hours, say 2 ounces for the first day, and add a similar quantity daily, until the limit is reached. Improvement sets

in promptly in about three-fourths of all cases; in the worst ones, where we must, as before stated, begin feeding with small quantities, progress is slow at first, but in any event we may expect the fall in weight to come to a sudden halt, to be presently followed by a slow gain. This gain, however, is partly due to water retention to make up for the severe water loss from the diarrhea; a real growth cannot be expected from albumin-milk alone, with its low caloric value. At the same time the fever rapidly abates, the stools speedily improve, gradually assuming the soapy quality characteristic of merely disturbed nutritional equilibrium.

As soon as the fever has gone down carbohydrates should gradually be added, 1 per cent. at a time. Birk⁶ was one of the first to insist on this; it should be done with caution, using the temperature, stools, and weight as guides. The temperature must remain at least nearly normal, the stools must not be diarrheal or badly digested, and the weight must show a tendency to be stationary, after the slight early gain, which has been shown to consist of water (Stolte¹). As soon as the baby has been brought back to its original condition of disturbed equilibrium the albumin-milk should be abandoned in favor of one of the regular formula mixtures (Finkelstein and Mever, 2 Stolte1). As to the carbohydrate to be added, the general consensus of opinion is in favor of malt rather than lactose or cane-sugar. In my own experience, albumin-milk plus malt has been a very satisfactory food for as long as six weeks, being given up chiefly because it was no longer necessary and troublesome to prepare.

The earliest American experiences with albuminmilk were not brilliant; Heiman² and Morse,⁴ while admitting that it might have a considerable field of usefulness, asked for more time and further trials before committing themselves in its favor. Finkelstein, however, and I think with some justice, criticises Morse's report as based on an incorrect administration of this food. Grulee² at once praised it enthusiastically, stating that he had given it continuously for months, by no means withdrawing it after a few weeks, as originally recommended. Friedländer and Greenbaum also had excellent results; they too kept it up for long periods, gradually running up the total sugar to 7 per cent. These reports agree fully with my own moderately extensive experience. I may mention that American reports are steadily growing more favorable, and that only a few foreign authors, like Cassel, are lukewarm, whereas a few French authors, e. g., Weill and Mouriquond, are almost alone in considering albumin-milk an utter failure.

Recently, Morse⁹ has observed that in cases of gas-bacillus infection malt is not so well tolerated as milk-sugar, being more readily fermented by this microörganism. It is quite possible that an accurate bacteriological diagnosis of the various types of infection may lead to a greater refinement in therapeutics, and Vincent makes a strong plea in this direction. It is, however, clear that, for the present, such methods are not within reach of the ordinary practitioner, who will still have to be guided by the symptomatology in treating his cases of infectious intestinal disease.

CHAPTER XI.

DISORDERS OF METABOLISM.

The disturbances of metabolism that are more or less in relation to infant feeding are quite numerous. They are mostly associated with artificial feeding, but may, under exceptional conditions, appear in nurslings; some few may be congenital, and in that event are more troublesome when the infants are bottle-fed. It is necessary for the practitioner to become intimately acquainted with these affections, as they are exceedingly common and must be reckoned with continually.

THE EXUDATIVE DIATHESIS.

Scrofulosis.—In the older works that deal with the diseases of children a great deal of space is devoted to the condition called scrofulosis. By scrofulosis was meant a diathesis characterized by enlargement of the lymph nodes and a chronic catarrhal condition of the mucous membranes, with a marked tendency to diseases of the skin, bones, and joints. The extremely chronic manifestations just referred to had a tendency to become acute or subacute from the most trivial causes, only to return, in the course of time, to their habitual state of a sluggish inflammation.

With the discovery of the tubercle bacillus a vast

amount of light was shed on the subject of scrofulosis. It soon was definitely ascertained that some scrofulous children were really tuberculous; not long after it was quite as clearly proved that others were not tuberculous. Thus, Cornet was enabled to classify all cases of scrofulosis as follows:

- 1. Tuberculous, caused by the tubercle bacillus.
- 2. Pyogenic, caused by pyogenic bacteria.
- 3. Mixed pyogenic and tuberculous infections.

For all practical purposes the first and third groups may be thrown together, leaving the second for further consideration. These last children presented somewhat of a pathological puzzle; they exhibited a constant liability to catarrhal inflammations, without any discoverable underlying cause, which gradually led to the chronic enlargement of one or more groups of lymph nodes, especially those of the faucial ring. Sometimes these children were described as affected with lymphatism, an unfortunate term that has been used to include a large number of conditions that may or may not be related.

About ten years ago a paper appeared, one of a series by Czerny,² in which he stated that these non-tuberculous, scrofulous children were the victims of a congenital and constitutional peculiarity which he called the exudative diathesis. The name was well chosen, as the most evident manifestation of this condition is a tendency to exudative inflammation of the skin as well as of the mucous membranes, the repetition of which results in the hypertrophy of the nearest lymph nodes.

269

Symptoms.—Czerny² describes the earliest stages of the exudative diathesis as follows: "Our suspicions should be aroused when apparently robust, but often more or less neurotic parents have a baby that seems puny and delicate; the first sign of trouble is failure to gain in weight even if supplied with breast-milk. The infant behaves almost as badly as if it were artificially nourished, even the human milk-fats being absorbed with some difficulty. The attending physician is very apt to accuse the maternal milk-supply of being at fault; we have, however, learned that a normal baby will do fairly well on rather inferior breast-milk, and it is important to bear this in mind when we have one of these cases before us. As a matter of fact the doubting attendant will promptly be undeceived if he resorts to a wet-nurse or to bottle-feeding; the former will do no good whatever and the latter will immediately lead to serious intestinal disturbance. Thus, feeding at the supposedly inferior maternal fount becomes imperative. As the infant grows older conditions improve a little, but the baby still lags behind the weight that is normal for its age; meanwhile, although there may have been no serious digestive disturbances, there is occasional feverishness, some anemia, a gradual or even rather rapid glandular enlargement, and often a tendency to develop eczema, all of which disorders are due to a feeble resisting power to mild infectious processes. Some of these infants are set back by frequent colds in the form of coryza, tonsillitis, or bronchitis; others manifest attacks of digestive disturbance; still others suffer

from a catarrhal condition of the genito-urinary mucous membrane, with a liability to cystitis and pyelocystitis (Lust, 5 Beck²); not a few are afflicted with several of these lesions. Most of the cases of infantile adenoids and tonsillar hypertrophy belong to this group."

Treatment.—It will be seen that it is necessary to keep these infants at the breast, at all hazards, and at that we must be careful about overfeeding. Any attempt to fatten these babies is not only useless but positively harmful; the four-hour nursing interval must be adhered to strictly. Even if we do succeed in adding a few pounds of weight, not very much is gained thereby, as fat infants who suffer from the exudative diathesis are just as much subject to complications as those of subnormal weight.

Artificial feeding of these infants is beset with the greatest difficulties, as the fat-digestion is exceptionally poor. Only low fat mixtures are tolerated at all, but even so, soap stools and obstinate constipation are the rule, and the replacement of milk-sugar with dextrin-maltose is usually necessary. In many cases bottle-feeding works out so badly that wet-nursing becomes imperative unless we wish to imperil the baby's life. Even the physiological process of dentition leads to irritability and functional disturbance; these are the babies that have created the popular apprehension of the dangers of teething, which never causes real illness in a normal infant. When the time for weaning arrives this transition is effected with difficulty, as by this time the baby is likely to have acquired some degree of

rickets and lymphatism; there is no little danger of a general breakdown at this period, and sometimes we encounter acute collapse and fatal heart-failure.

Shortly after Czerny proposed this clinical entity numerous objections were raised; it was declared that he had merely been describing lymphatism under a new name. In a later paper Czerny⁴ answers this criticism by explaining that the exudative diathesis is a congenital predisposition to infection, whereas lymphatism is an acquired condition, often, to be sure, the result of the former disease. He improves the opportunity to go more fully into the subject of overfeeding, so widely practised in bringing up infants, either naturally or artificially. He points out that whereas some babies lay on fat easily, if they are overfed, and seem none the worse for it, others simply cannot be fattened. For this peculiarity the following reasons may be held accountable: either the child is partaking of an hereditary tendency to leanness, in which case it will be doing quite well in every other respect, or it will not gain because of poor absorptive faculties, in which case it belongs to the class here under discussion.

Czerny's views have not yet gained general acceptance, but have a great deal in their favor. They offer the only explanation, hitherto afforded, for those children that will not thrive on any sort of feeding, including the breast, and account for the development of many cases of rickets and lymphatism in breast-fed babies who have been brought up under the best hygienic conditions. Much of the objection is directed

at the term chosen by Czerny; this point is hardly worthy of discussion; future observations will finally decide if the exudative diathesis is to be regarded as a clinical entity.

ANEMIA.

We have seen that the supply of iron is scarcely adequate even in human milk; many breast-fed babies, who are otherwise doing fairly well, have a hemoglobin index of barely 80. In artificial feeding matters are worse, for cow's milk contains relatively little iron, and the absorption of that metal, going hand in hand with the deficient absorption of calcium, is hardly ever equal to the baby's requirements; an index below 70 or even 60 is therefore not uncommon in bottle-fed infants.

It is characteristic of infantile anemias that they take on severe and apparently even pernicious forms on occasions that, in later childhood, produce much milder manifestations. Morse⁵ has shown that the type first described by v. Jaksch as anemia pseudoleukæmica infantum, with marked lymphocytosis, myelocytosis, splenic enlargement, nucleated and degenerated red blood cells, and an extremely low hemoglobin content, is nothing but the severe phases of the ordinary secondary anemias at that age. Koplik⁴ has shown that v. Jaksch's anemia is not infrequently of intestinal origin; he reports a number of cases in infants suffering merely from a prolonged condition of disturbed equilibrium, which we know to be charac-

ANEMIA 273

terized by specially poor assimilation of calcium and iron. Finkelstein⁸ gives an instructive review of this subject, stating that the regular cause of infantile anemia is an insufficient absorption of iron from the food.

Treatment.—Finkelstein's plan of treatment consists chiefly in dietetic measures; he gives cereals, purées of vegetables that contain much iron, such as spinach and carrots, and also fruit juices. By way of medication he gives iron and occasionally minute doses of arsenic. Of the iron preparations he rightly considers the inorganic preparations quite as useful as the modern proprietary organic compounds; my personal preference is for the syrup of the iodid of iron, in doses of 5 to 10 drops, t. i. d., in sweetened water. Finkelstein also considers the climatic treatment of infantile anemia, and does not agree with those who recommend high altitudes; he prefers slight elevations up to about 1200 feet, for dwellers near the sea-level. It should be remembered that the rarefaction of the air, at the elevation of a mile, as for example at Denver, Colorado, at once creates a requirement of an additional 30 per cent. of red blood cells and hemoglobin, thus imposing an extra strain on an organism that is already deficient in both these elements. I have gone into this matter quite fully in another place,3 and at this point shall merely state my entire agreement with Finkelstein's conclusion.

It is, of course, understood that, whenever possible, the anemia of infants should be treated by removing the cause, that is, by correcting any palpable errors in the diet. A general examination is likely to reveal other physical deficiencies, particularly rickets, of which the anemia is often merely one symptom.

ACIDOSIS.

By acidosis we understand an abnormal form of metabolism, of a very obscure nature, which results in the formation of certain low fatty acids, particularly β -oxybutyric acid, and manifests itself most obviously by the appearance of diacetic acid and acetone in the urine, as well as by certain constitutional symptoms of intoxication. The normal alkalinity of the tissues is diminished, and the ammonia that should neutralize the mentioned acids is eliminated as such. The classical picture of acidosis as it occurs in patients suffering from diabetes mellitus is clearly delineated by Naunyn as well as Chittenden and Mendel. Acidosis occurs also under other conditions. though usually in a less severe degree, and is not uncommon in young children and even infants. Its etiology in early life is not altogether unrelated to its causation in diabetes, so that we may safely refer to the authorities just mentioned in studying this metabolic error as found in infancy.

Acidosis occurs, when the organism is not assimilating a sufficient amount of carbohydrates or fats, either through a pathological inability to utilize them, after absorption, as in diabetes mellitus, or because of an insufficient intake. As is well known, both causes often work together in the deadly form of acidosis

occurring in diabetics. In childhood it has generally been rather hastily assumed that acid intoxication is due to overfeeding with carbohydrates; the greater probability that carbohydrate starvation is the cause has hardly been regarded seriously. Either cause, as well as fat starvation, may be operative in producing this condition in infancy.

When too little carbohydrate and fat is assimilated from either cause, the body fats, secondarily the proteins also, begin to be disintegrated, not as normally, chiefly into water and carbon dioxid, nor in normal amount, but largely into intermediate bodies of the fatty-acid group and in great quantity. Meanwhile, as stated, the ammonia that should neutralize these acids fails to do so for reasons hitherto obscure. The pathological effects of the acids are still in doubt; we cannot, for the present, say whether they are injurious because of the reduced alkalinity of the tissues, or if they are directly toxic; probably both aspects of this matter are of importance.

As there is always some breaking down of the body fats, and as the neutralization by ammonia is not absolutely to be depended on, a slight and occasional excretion of acetone in the urine is hardly to be regarded as pathological, and may occur in normal infants (Baginsky²). It is, however, rare under physiological conditions, and Schrack claims that it is never found in babies fed on a pure milk diet. Little of interest, from the stand-point of infant feeding, was added to our knowledge until, some years ago, Marfan² reported a series of cases in children in whom

acetonuria was associated with recurrent or periodic vomiting. In this condition it was not a question of traces of acetone but of such large quantities as half a gram per liter. Soon after, Edsall published an account of a similar group of cases, and since then reports have been numerous, so that this clinical picture has become familiar to all of us. In 1905 two interesting papers, giving a description of the same syndrome in infants, were published by Morse⁶ and Langstein and Meyer. In both it was shown that acidosis is not rare in infants who suffer from gastro-intestinal diseases, which is a much less frequent association in older children and adults. Furthermore, evidence was introduced to show that the acetonuria was due to carbohydrate starvation by itself or as part of a general starvation, and that alkali loss, through an excess of low fatty acids in the intestine, was a regular feature, so that acidosis was brought into close relation to our old enemy, the poor absorption and assimilation of cow's milk.

Since then comparatively little has been added to our knowledge of this condition. It is not particularly frequent in infants because the error of carbohydrate starvation is rarely committed at that age; most of the cases occur in relation to starch-atrophy, and here the course of events is somewhat different. In starch-atrophy (Mehlnährschaden) we have two factors operating: (1) a breaking down of the carbohydrate assimilation, from persistent overloading with largely unassimilable starch, reminding us of the conditions in diabetes; (2) the infant that is brought up on one of

the starchy baby-foods is subjected to a persistent fat starvation which results in an excessive breaking down of the body fats, largely into substances of the acetone group. Frew notes the interesting circumstance that slight acidosis often occurs when a baby is transferred from the breast to the bottle, and attributes it to carbohydrate starvation; the probability is much stronger that the difficult process of learning to absorb the cow-milk fats and temporary fat starvation play at least an equal role.

Treatment.—As in diabetics so in infants, we may treat acidosis along two very different lines; we may give alkalies or we may add carbohydrates and fat to the diet. As in diabetic acidosis, so here also the administration of alkalies affords only symptomatic treatment, and in no wise affects the underlying cause. At that the fixed alkalies are not an exact substitute for the body ammonia, which should effect the neutralization of the acids, and, if given in sufficient quantity, they interfere seriously with the gastric functions. Sodium bicarbonate is the favorite alkali for this purpose, but is likely to do as much harm as good; we have seen the objections to loading up the infant organism with an excess of sodium. Potassium bicarbonate, given in a dosage not exceeding three grams (45 grains) per day, to a ten-pound infant, would theoretically seem far preferable.

It is more rational to seek the removal of the cause of acidosis, which may be accomplished by a combination of two measures. First, the sugar should be run up to the normal ratio of 7 per cent.; secondly,

we must see that the other food constituents are presented in sufficient quantity, at the same time looking to adequate absorption of the fats; this last may be aided by giving malt, instead of milk sugar, if the stools furnish evidence of deficient fat digestion. In any case, acidosis, in infants, should be very amenable to treatment, since it can occur only when the diet scheme is very faulty or deficient.

THE METABOLIC DERMATOSES.

Eczema.—A whole school of dermatologists maintains that eczema is a cutaneous disease, pure and simple, and in no sense merely symptomatic of an internal disorder; this stand-point does not always hold good for adults, and is certainly not true for all children. In infants most, if not all, cases of eczema are in some relation to disturbed metabolism, and the circumstance, that we cannot always indicate the latter with precision, is not sufficient to invalidate the general proposition.

Eczema is especially common in infants that suffer from the exudative diathesis, in fact, this constitutional infirmity derives its name from the tendency to exudative inflammatory processes, of which eczema is one of the most conspicuous. Moro and Kolb have furnished an especially exhaustive study of eczema, in which some interesting points are elaborated. For some unknown reason, it is commoner in boys; the infants are usually bottle-fed and fat; they have, in addition to the eczema, a marked tendency to

urticaria, and are often affected with rickets and spasmophilia. The last point is not remarkable, in view of the predominance of artificial feeding. Moro and Kolb do not hesitate to say that the exudative diathesis, or in other words, the congenital tendency to eczema, is the most essential etiological factor, aided of course, in many instances, by external injurious influences, such as heat, cold, moisture, and irritating agencies of various sorts. Holt¹ is quite sure that high fat feeding is in part responsible; other authors accuse the whey salts, and recommend a whey-free food, such as albumin-milk plus sugar (Finkelstein³); Bruck, however, succeeds quite well in eliminating whey as a factor.

There is an old superstition, still prevalent, that the endeavor to cure infantile eczema is apt to lead to dangerous and even fatal consequences: the disease is popularly said to strike inward. It is a fact that eczematous babies sometimes die suddenly, from no discoverable cause; such cases, as the one reported by Hichens, in which sudden death promptly followed a cure of the eczema, are not calculated to reassure the lay mind, though similar fatalities have been noted in unsuccessfully treated cases. Feer² has discussed this matter very thoroughly. In medical circles, these deaths have been generally attributed, rather vaguely, to the condition called lymphatism; it is more accurate to regard them as due to spasmophilia, which is not rarely fatal, and is a common complication of the exudative diathesis. study of the case reports shows that the cure or continuance of the dermatosis has nothing to do with these so-called eczema-deaths.

Treatment.—As to the treatment of these cases, aside from local applications, which are always necessary, and sometimes successful by themselves, we can say little that is definite. Our dietetic treatment of infantile eczema is unsatisfactory, and decidedly empirical, because we cannot always lay our fingers on the dietetic error present in the individual case. In addition, we are confronted with the far from easy task of combating the exudative diathesis, the cause of the frequent chronicity of this affection, so that the problem becomes doubly difficult.

Nevertheless, we can take certain dietetic precautions, which may help us to our goal. The fats should, at any rate, be kept within reasonable limits, and we can easily avoid an excess in carbohydrates and salts, but more definite recommendations, as to other modifications, cannot be given. Each case must be considered separately, and some cases resist all dietetic treatment indefinitely, finally being cured by local measures alone.

Finkelstein¹⁰ warns us against a routine starvation treatment of infantile eczema; this is indicated only in cases of obvious overfeeding, and usually modification is more desirable than a general reduction of the food allowance.

Urticaria.—Urticaria is the metabolic dermatosis par excellence; it is very common and obstinate in infancy, occurring either in the typical form of wheals, or in the papular form, called strophulus or

lichen urticatus. In many cases, but not so often in this country, it represents the early stage of the constitutional affection known as prurigo. Before making the diagnosis of endogenous urticaria, the practitioner must first assure himself, in the individual case, that biting insects have nothing to do with the eruption; many cases of supposed urticaria are due to bugs, mosquitoes and the like.

Recent studies on anaphylaxis seem to demonstrate that the ingestion of certain foreign proteins is the cause of urticaria, though other factors may sometimes be responsible. According to this theory, the proteins of human milk should never cause urticaria. cow-milk proteins only under exceptional conditions, and such altogether foreign bodies as eggs and vegetable proteins more frequently. The observed facts are in accord with this hypothesis, to a certain extent, but many points cannot as yet be considered proved. As to the toxicity of egg-albumen for certain individuals, and referring to observations in infants only, I merely cite the experiences of Baginsky and Bendix;² O. M. Schloss has made a thorough study of the subject, and established the presence of an anaphylactic reaction for at least some cases. Among offending substances, other than eggs, may be mentioned almonds, oatmeal, besides several products of protein decomposition, such as the metabolic products of intestinal parasites, and certain substances formed in imperfectly preserved foods; the urticaria, that often follows the hypodermic injection of horse-serum, is familiar to all.

As to the production of urticaria by bodies, other

than proteins or protein derivatives, we cannot, for the present, speak positively, but all the available evidence is against it.

Treatment.—The successful treatment of urticaria, especially in infants, has been hampered by our imperfect etiological information, and by difficulties in removing the cause. After we have eliminated such factors as insect bites and intestinal worms, we should direct our investigations to the ingested proteins; if cereals have been given, they should be stopped, oatmeal especially having recently become the object of considerable suspicion. Some authors suspect the whey-proteins of cow's milk as being more foreign than the casein; on this hypothesis we are justified in going over to sweetened albumin-milk. Now and then, we may encounter one of the cases of cow-milk idiosyncrasy, previously described; in that case, breast-feeding is the only remedy.

Many authors, who have not convinced themselves of the anaphylactic nature of urticaria, hold the carbohydrates responsible; as to this, I can only repeat that this theory has not a shred of real evidence to sustain it.

Preliminary to dietetic and medicinal measures, a brisk purgation with castor oil is the most rational treatment, for thus at least the intestinal tract is cleared of toxic substances. At the same time, we may treat the intense itching symptomatically, with soothing applications such as talcum and menthol. The alkalies have been widely recommended, largely on empirical grounds, for there is no evidence that

acidosis plays any role in urticaria. If we give limewater, sodium bicarbonate, or sodium citrate, it should be in small doses, so as not to interfere with the gastric functions. Treatment with calcium salts, especially the lactate, has often been advised. The rationale of this procedure is far from clear, being based on the supposed relation of urticaria to certain hemorrhagic diseases. I have given this treatment a fair and quite extensive trial, and can state very positively that it has yielded me no results whatever. The best plan, so far as I can see, is to experiment with protein withdrawal, eliminating one possible offender after another, until we find that the cutting out of some one albuminous food causes cessation of the disease.

CHAPTER XII.

RICKETS AND SCURVY.

RICKETS.

RICKETS is a chronic impairment of nutrition, formerly regarded as affecting chiefly the bones, but now recognized as implicating all the tissues of the body, including particularly the nervous system. It is exclusively a disorder of development, and therefore peculiar to infancy and childhood, almost always beginning in the first year of life.

Etiology.—The etiology of rickets is essentially dietetic. Other hygienic factors play an auxiliary and predisposing part, but the chief cause is artificial feeding. In our large cities, especially among negroes and Italians, moderate rickets may often be observed in breast-fed infants, but, generally speaking, the severe types of this affection are met with only in bottle-fed babies. The influence of bad general hygiene cannot, however, be ignored; Hansemann¹ lays great stress on the lack of fresh air as a predisposing factor, and considers the keeping of infants indoors as effective as unsuitable food in the production of rickets. These remarks are in accord with the racial observation just made; negroes and Italians

dread our severe winter climate, and do not take their babies out of doors in cold weather as freely as they should. I have been able to verify this statement in my dispensary service, where about one-tenth of the breast-fed infants present the symptoms of mild rickets; inquiry has shown that many of these babies have been kept home a great deal, the mothers not being able to spare the time to take them out into the air. Nevertheless the proportion of rickets is incalculably greater among bottle-fed infants, nearly all of whom are at least slightly affected, even in well-todo families, where the lack of fresh air is out of the question; it is also very common in bottle-fed country children. Furthermore, the severe forms are almost exclusively found among the artificially fed. The net result therefore is that bottle-feeding is the vastly predominating cause, which may indeed be aided by defective general hygiene, but is quite effective without it.

To comprehend the dietetic causes of rickets we must go back to first principles. We have seen how poor the absorption of the fats of cow's milk is under the most favorable conditions and how the absorption of calcium and phosphorus suffers concurrently; we have also seen how much of the calcium goes to waste after absorption. The effect of this is most readily observed in the bones, the chief depositories of calcium and phosphorus; however, the nervous system also requires a large amount of phosphorus for its proper development, and a certain quantity is needed for the remaining tissues. The sum of

these observations is that rickets is the manifestation of calcium and phosphorus starvation, caused by the imperfect absorption of the fats from cow's milk; we see at once why rickets is so much less likely to appear in breast-fed infants even under adverse hygienic conditions.

It is important to remember that the ability of the bones and other tissues to take up calcium and phosphorus is not in any way impaired; the trouble lies with the insufficient supply of these elements, and rickets has been caused in animals by simple deprivation of these substances. There is a definite minimum below which the tissues suffer from malnutrition; Aron estimates that the infant should add on 12 grams of lime for every kilogram of total gain in weight.

Deficient fat absorption is by no means the only factor in producing rickets; in the past the unwarranted fear of casein also played an important part. Since the food phosphorus is wholly or almost entirely derived from casein, it is quite evident that the development of rickets may be favored by protein starvation; this factor will probably become less influential with the abandonment of the fears as to the indigestibility of casein. I hope that the reader will not be troubled by the apparent paradox that low fat-feeding alone does not cause rickets even if carried out to an excessive degree; we have seen that when high fat ratios are given much of the fat is wasted: it is the waste fat that binds the calcium and renders it unavailable, but a shortage of fat has no effect on the calcium metabolism. Fat-starvation may cause other disturbances, notably acid intoxication, but never rickets.

Symptoms.—While rickets affects all the tissues of the body the most conspicuous lesions are in the bones, which present a condition that has been variously interpreted as chronic inflammation (Kassowitz³) or chronic malnutrition. As is well known, the growth of bone takes place under the periosteum and at the epiphyseal lines. In rickets we find the latter swollen and hyperemic, with increased vascularization, finally leading to a marked palpable and even visible thickening of the ends of the long bones; at the same time there is a conspicuous deficiency in the deposition of true osseous tissue, because the necessary calcium and phosphorus are lacking. Pfaundler³ declares the process to consist in the inhibition of the normal biochemical changes in the tissues, but this merely states a fact; it does not explain how the absence of calcium and phosphorus causes the local manifestations that are decidedly of an irritative type. The epiphyseal hyperemia must be either compensatory or due to the stimulus of a chemical body, possibly sodium chlorid, that is taking the place of the normal calcium and phosphorus.

Concurrently with the lesion at the epiphyses the subperiosteal development of the bones is likewise impaired, and the cortex is also deficient in calcium phosphate, so that the shafts are much softer and more yielding than normal; the same is true of the cranial bones.

The objective consequences of these defects are

as follows. The long bones are thickened at their ends and their shafts bend easily, so as to be subject to permanent curvatures and green-stick fractures. The ribs are beaded at the junctions of the costal cartilages, forming the so-called rosary, and the ribs themselves are drawn inward by the traction of the diaphragm, causing the development of a groove along the insertion of that muscle (Harrison's groove) and a protrusion of the sternum (pigeon-breast). When the infant begins to stand up and walk, bow-legs, knockknees, and flat-foot develop, The cranial bones are often so badly ossified as to bend like paper under the pressure of the finger (craniotabes); the closure of the fontanelles is much retarded, the head gradually assumes a squarish form, and occasionally becomes somewhat enlarged from a slight degree of hydrocephalus, to be again referred to later. This enlargement of the head persists as more or less marked and permanent bulging into later life.

The pelvic bones sag as soon as the infant begins to sit up, and undergo a deformation that has serious consequences in females in the child-bearing period; this defect escapes notice in infancy, but is none the less to be borne in mind as a modifying factor in prognosis and as an indication to treat the disease early and thoroughly.

These infants are late in sitting up, standing, and walking, not only because of weakness of the bones, but still more on account of their poor muscular development. The weakness of the dorsal muscles leads to the frequently observed pseudokyphosis that

is characteristic of rickets and must be carefully differentiated from the bony kyphosis of Pott's disease. The visceral unstriped muscles are also underdeveloped, so that the motor functions of the digestive tract are performed very inefficiently; there is a marked tendency to enteroptosis, and always more or less intractable constipation. The pot-belly that is so characteristic of rickets is due to relaxation of both the visceral and abdominal muscles.

Rickets more or less affects all the internal organs. Enlargement of the spleen is almost constant, of the liver usual and of the lymph nodes very common. The visceral mucous membranes are somewhat hyperemic and subject to catarrhal inflammation; there is, therefore, a considerable liability to bronchitis and enteritis, reminding one of the exudative diathesis. The subcutaneous fat may be normally and even excessively developed, as the carbohydrate assimilation is perfectly intact and likely to supply this demand quite adequately. The dentition is tardy and often irregular, and the teeth are apt to be ill-formed and excessively subject to caries. The blood always shows a fairly severe anemia, as the calcium and iron deficits go hand in hand. The nervous system is very deeply involved, presenting the condition that we shall consider later under the head of Spasmophilia.

General sweating, often profuse, is characteristic of rickets; it is partly due to the general condition, for the nutrition of the skin is also impaired, though this is not demonstrable, and partly to the local bone hyperemias, being particularly intense about the

head, even in the absence of noticeable craniotabes, and also very marked about the epiphyses. The sweating of the scalp leads to restlessness, and the formation of a bald patch on the occiput, which is almost pathognomonic of rickets. The increased perspiration is not associated with a rise of temperature.

We must take care to remember that the lesions of rickets are so chronic as to be absolutely painless. Tenderness of thickened joints should lead the attendant to look for scurvy and not rickets.

Diagnosis.—The diagnosis of rickets is usually exceedingly easy; scurvy is the only affection that somewhat resembles it, but scurvy is an acute disease, with extreme local tenderness as one of the cardinal symptoms; what was formerly called acute rickets is now known to be scurvy. Occasionally the peculiar arrest in development called achondroplasia is designated as fetal rickets; it has nothing whatever to do with rickets, not being in any respect a disease of nutrition, but resulting from a dysplasia of the mesoblast in embryonic life. There has been considerable discussion as to the occurrence of true congenital rickets, with the preponderating evidence on the affirmative side; there is no doubt, however, of its extreme rarity in this country, and foreign authorities are gradually coming around to the opinion of Hansemann² that it must be exceedingly infrequent anywhere.

The tendency of rachitic infants to inflammatory processes furnishes a point of contact with the exudative diathesis, and it is highly probable that many reports of congenital rickets refer to the latter affection.

The lines of divergence are, however, numerous. Rickets is associated almost exclusively with artificial feeding, the bone symptoms occur in no other disease, there is no tendency to be subnormal in weight, and we often observe the nervous symptoms to be detailed presently. The exudative diathesis is congenital, persists in spite of breast-feeding and perfect hygiene, is almost always associated with a marked deficiency in weight, and entirely lacks the characteristic bone lesions of rickets. Of course, an infant suffering from the exudative diathesis may acquire rickets, and is pretty sure to do so if fed on cow's milk; in that case there will be no occasion for a differential diagnosis.

Prognosis.—Since rachitic babies are very liable to inflammatory diseases of the mucous membranes, and are often affected by the dangerous nervous complications presently to be described, the prognosis as to life is seriously impaired as compared with normal infants. The prognosis as to duration is not at all good, for years are consumed in the functional compensation of the rachitic after-effects, which are always more or less permanent, and rickets of moderate severity never fails to pass on ineffaceable stigmata into adult life.

Treatment.—All that the treatment of rickets can do is to arrest the progress of the disease. It goes without saying that a good general hygiene is essential. Fresh air comes first, because of its favorable influence on metabolism; the infant should spend most of the day out of doors except when it is actually raining; mere cold should be entirely disregarded. The bedroom

should be well ventilated, and the employment of a hair pillow, instead of a feather pillow, tends to check sweating of the scalp, as moderate lightness of the bedclothes tends to check general sweating. A lukewarm bath should be given daily, followed by cool sponging; rachitic babies do not tolerate cold bathing, which should never be attempted. When the circumstances of the family permit, the infant should be taken into the country, as rickets is, to a very large extent, an urban disease.

Breast-feeding, while not by itself an absolute preventive, is nevertheless unfavorable to the development of rickets, and should be resorted to whenever possible; failing this recourse we must remedy any demonstrable errors in the scheme of artificial feeding. It is not only useless but positively harmful to run up the fat percentage; the fats are already poorly absorbed, and it is rather advisable to cut down this element in the food until the stools become approximately normal. On the other hand we can often benefit the infant by running up the casein as high as possible, for thus a good supply of available phosphorus is assured. From this we may readily conclude, that the most advantageous diet consists in a whole-milk mixture, in adding to the concentration of which we raise the protein ratio concurrently with the fats: we can increase the absorption of the latter by using malt instead of cane-sugar or lactose. Attentive observation of the stools is necessary, for soap stools show a heavy loss of calcium, which is inconsistent with improvement in the rachitic condition. Constipation is always a troublesome feature, aggravated by its dependence on two concurrent factors, namely, the fat-indigestion, and the poor muscular development of the intestine. One way of combating this symptom is through the substitution of oatmeal water for barley-water as the milk diluent; another consists in giving small doses of magnesium peroxid (milk of magnesia), but I have found this only moderately effective; in many cases we must be content to put up with a certain degree of constipation in the earlier period of treatment, relieving it with enemas or suppositories when necessary.

The medicinal treatment of rickets has undergone great vicissitudes. Setting out from the pathological basis of this disease we ought to secure improvement by giving phosphorus and calcium, but the effective administration of these substances is not so simple as it would seem; their absorption and assimilation are subject to certain biological limitations that are not, at first sight, obvious, but have been proved experimentally and must be reckoned with. phorus, given in doses of a fraction of a milligram in oily solution, was first recommended by Kassowitz,¹ and seemed to produce fair but slow results, which were only tolerably satisfactory, though we now know that very rapid progress cannot be expected. Common white phosphorus is, moreover, a somewhat dangerous drug unless given with great caution; attempts were therefore made to substitute harmless phosphorus compounds as safer and perhaps equally absorbable and assimilable. These endeavors only

served to discredit phosphorus therapy; the effects of the inorganic phosphorus compounds on rickets are very doubtful, though a few recent investigators assign some value to them; these doubts also apply to the vaunted glycerophosphates, as well as most of the modern and complicated phosphorus preparations, which are quite as inert as the ordinary phosphates and hypophosphites. It is probable that the body can absorb phosphorus only as such or by splitting such organic phosphides as casein.

Treatment with calcium salts has been tried extensively, and seems, at first sight, most rational; in practice, however, it is ineffective unless phosphorus is given at the same time, and even then its necessity is doubtful. Although the calcium waste appears to be the essential factor in producing rickets, it is better to control the loss of that element by giving phosphorus than to add lime-salts to a diet that already contains more than sufficient calcium. Schloss and Frank report increased retention of calcium and phosphorus from the administration of calcium phosphate, but only if cod-liver oil is also given, so that the role of the former is more than doubtful. Schabad has shown that the administration of pure phosphorus in cod-liver oil triples the calcium assimilation in rickets, but is without effect in normal infants; he attributes much of the success to the oil, observing that cod-liver oil is a particularly absorbable fat which binds the calcium to a less degree than do the cow-milk fats: therefore by giving phosphorus in this fashion we both supply that element and favor the assimilation of calcium, more of which is left free to be deposited in the tissues of the body.

From the above we may fairly conclude that any measure that promotes the absorption of fat is likely to give us the same result. The practice, now growing very popular, of giving pure phosphorus in doses of about $\frac{1}{200}$ grain (0.3 milligram) in malt-extract seems therefore perfectly sound, as malt also favors fat absorption, thus diminishing the calcium waste. Both cod-liver oil and malt as vehicles of phosphorus seem to hasten its curative action in rickets; both methods can easily be controlled in the individual case by watching the feces. If these tend to become normal and the constipation ceases we may be reasonably certain that the progress of the disease has been arrested.

If we have, provisionally, passed over the neurological manifestations of rickets it is because they have been generally treated as a distinct disease. A change in opinion is now in progress, but no harm will be done by discussing this group of lesions separately under the head of

SPASMOPHILIA (TETANY).

The terms spasmophilia and tetany are often employed interchangeably, but are by no means synonymous. Tetany is a condition of muscular spasticity (hypertony) that may arise from the most varying causes; it is one of the symptoms of a variety of conditions, including such diverse diseases as tetanus and strychnin poisoning. Spasmophilia is the better

name for the condition before us, as it designates a diathesis characterized by a tendency to tetany which may, however, be latent or temporarily in abeyance.

By infantile spasmophilia we designate a liability to tonic and clonic spasms of every kind occurring in early life; the field is thus evidently a wide one, including the most various clinical manifestations: (1) we recognize clonic convulsions of the eclamptic type, occurring without any evidence of intracranial, visceral, or acute febrile disease; (2) we note the tonic spasms or contractures that are characteristic of what is properly called tetany; as well as the cataleptic seizures described by Epstein; (3) there is the classical triad of symptoms: Chvostek's, consisting in reflex overexcitability in response to tapping of the muscles, best shown in the face; Trousseau's, consisting in tonic spasm of a slightly constricted extremity; Erb's, consisting in increased reaction to electric currents. Recently a number of other symptoms have been assigned to spasmophilia. Escherich³ claimed that laryngospasm was a part of this clinical picture, and his view was only apparently contradicted, but really confirmed, by Kassowitz,2 who considered this symptom a sign of rickets. Hochsinger furnished convincing evidence that at least a majority of the cases of infantile nystagmus and nodding spasm, which form a syndrome, belong to this group. Not without interest is a recent statement by Hess⁵ in which certain cases of pylorospasm, cardiospasm, and pharyngospasm are regarded as phases of spasmophilia. Careful examination of a large number of infants has shown that there are many cases of latent spasmophilia in which we can find only one or two symptoms of the Chvostek-Trousseau-Erb triad, without the presence of any of the severer manifestations; it is worthy of note that these infants are regularly rachitic.

The very circumstance that we find rickets in practically all the cases of spasmophilia would lead us to conclude that the latter is merely a manifestation of the former; for many years, however, exact proofs were lacking. At one time considerable doubt was cast on this relationship by the discovery that tetany was caused by removal, or disease of, the parathyreoid glands. Stöltzner, however, showed that, aside from the spastic symptoms, spasmophilia parathyreopriva and spasmophilia rachitica have very little in common; the latter presents neither tremor nor fibrillary twitching nor tachycardia nor stupor; it is made worse by continued feeding with cow's milk, whereas the former is uninfluenced by the diet: Stöltzner errs, however, in considering spasmophilia a manifestation of calcium poisoning; we now know that rickets is the result of calcium-starvation, and Nothmann³ has shown that the parathyreoid form of spasmophilia is also associated with a calcium deficit. This point forms indeed the one connecting link between these two affections; both are associated with a lack of calcium, but their ultimate causes are quite different, from the evidence now at hand.

Schabad³ shows that the metabolism of spasmophilia is identical with that of rickets; Cybulski has demonstrated that the spasms disappear as the calcium

retention improves; finally, Curschmann reports two cases of fatal spasmophilia in which the parathyreoid bodies were found normal on autopsy.

Symptoms.—The reader will recall that in the discussion on starch-atrophy (Mehlnährschaden) we noted muscular rigidity as a conspicuous symptom in the later stages; in other words, the infants affected with malnutrition from this cause exhibit signs of spasmophilia. Ackermann takes Czerny and Keller to task for attributing this condition to the ingestion of excessive amounts of starch, and so far is justified in his criticism. Nevertheless the clinical observations of Czerny and Keller were quite correct insofar as hypertony of the muscles does occur as they describe; the only real question involved is that of causation, and that is capable of explanation. The trouble with feeding on Nestlé's food and the like is not only because of the excess of starch, but even more on account of the attendant protein starvation. Starch-atrophy is a complicated process in which carbohydrate intoxication plays at the most a terminal role, fat-starvation and acidosis are conspicuous, as we have seen, and rickets, including spasmophilia, an essential feature. Keller's treatment recognizes all these characteristics; it does not so much cut down the quantity of the carbohydrates as substitute more digestible ones; it gives some fat and renders it more assimilable by also giving malt; it further checks the calcium waste, through the addition of potassium carbonate, which distinctly favors calcium retention. Thus, Keller's methods are based on a correct clinical observation, and work toward the desired end, even though all our present biochemical data were not available a decade ago when this plan of feeding was devised.

The observation of Lust⁶ that potassium, given as a chlorid, is spasmogenic, cannot for the present be reconciled with the above; we do well, however, to recall that spasmophilia was regarded as due to calcium-poisoning by Stöltzner, whereas Lust's researches indicate that the administration of this element reduces the motor excitability. This question is evidently still unsettled.

I think that I have presented sufficient evidence of the close interrelation of rickets and spasmophilia; such recent authors as Langstein³ commit themselves unreservedly to this proposition. We shall probably not go far astray if we regard spasmophilia as the manifestation of rickets in the nervous system, and its cerebral symptoms as due to meningeal hyperemia in connection with cranial rachitis. This theory receives support from the presence of slight hydrocephalus in severe cases. It only remains for us to clear up some points of differential diagnosis. In the first place we must not confuse larvngospasm (larvngismus stridulus) with congenital larvngeal stridor; the former consists of a true spasmodic closure of the rima glottidis, lasting a minute or thereabouts, and ending in a deep sighing or crowing inspiration; congenital laryngeal stridor is more or less constant and does not occur in fits. The former is not congenital but begins between the third and twelfth months, the very period when

the underlying disease, namely rickets, begins to show its characteristic development. General convulsions must also be subjected to a careful differentiation; the spasms of genuine spasmophilia are apt to occur without any demonstrable cause, whereas in other infants convulsions are either of distinctly cerebral origin or mark the beginning of a sharp rise of temperature, corresponding to the chills of later life. The nystagmus must be distinguished from that due to labyrinthine disease; this is often exceedingly difficult, as the precise mechanism of spasmophilic nystagmus is still unsettled, the prevailing view being, that it is merely the consequence of weakness of the motor apparatus of the eye-ball; examination of the ears will naturally clear up many doubtful cases. I cannot subscribe to the proposition, made in some quarters, that a slight Chvostek phenomenon may be disregarded if other spastic signs are wanting; it seems safer to keep in mind the picture described as latent spasmophilia, especially in view of the fact that practically all of the infants thus affected present symptoms of typical rickets.

Prognosis.—The prognosis of spasmophilia is very doubtful, as the general convulsions not uncommonly prove fatal; laryngospasm is also a threatening symptom, and the seriousness of an infection of these babies with respiratory disease or whooping cough is readily understood and can hardly be overestimated. Whooping cough alone is responsible for hundreds of deaths annually in the city of New York, and this mortality is chiefly among rachitic and spasmophilic infants.

Treatment.—The treatment is that of the underlying rickets. Calcium salts have been tried extensively for the attacks themselves. Freudenberg and Klocmann observe that their action is prompt but only temporary; Blühdorn gives calcium chlorid and bromid in daily doses of 2 or 3 grams, but claims only symptomatic value for his treatment; Aschenheim gives 7 to 10 grams daily of calcium lactate. The reason for the mere transitoriness of the action of calcium is the same as in the case of the ordinary symptoms of rickets; the calcium is not permanently assimilated. I must not fail to repeat that many authors consider calcium spasmogenic, so that this course of treatment still rests on an insecure foundation. Berend found magnesium salts valuable, again in opposition to the prevailing views, though the investigations of Meltzer and Auer have probably settled conclusively the usefulness of magnesium sulfate injections in the spasms of true tetanus.

More satisfactory are the results obtained from the treatment of the underlying disease, which has already been discussed. The symptomatic treatment, described above, may be employed during the time that we are gradually correcting the general nutrition, which always takes some weeks, at least, before improvement is apparent. Spasmophilia is by far the most serious manifestation of rickets, and the chief reason for energetic prophylactic measures against this disease, which, to a decided degree, is to be classed among the preventable disorders, its present marked prevalence not being creditable to

modern pediatrics. On the other hand it cannot be denied that something has been accomplished in this direction; with the growing insistence on breast-feeding, the steadily improving methods of artificial feeding, and the general rise of hygienic standards it is certainly true that the worst forms of rickets are gradually disappearing from our hospitals, so that recent decades have shown some little progress.

SCURVY.

The occurrence of scurvy, in infants, was first suggested by Glisson, but its general recognition dates from an article by Möller, in which he discussed what he called acute rickets. Jalland was the first modern writer to identify infantile scurvy, antedating the classical report of Barlow ten years. The general acceptance of this identification with the scurvy of adults dates from the closing years of the last century.

Etiology.—As in adult, so in infantile scurvy, the precise etiology is still uncertain; we know that it follows in the wake of a diet consisting too exclusively of certain preserved foods, and conversely, that fresh and especially raw foodstuffs of various kinds absolutely prevent its development, besides affording a specific cure. On this subject the most comprehensive investigation has been made by the American Pediatric Society, of which a good summary is given by Griffith; it shows that the majority of infants who are affected by scurvy have been fed on

proprietary baby-foods, and that the employment of condensed and sterilized milk accounts for most of the remainder. The observation that a very few cases have been fed at the breast, or on raw cow's milk, must be interpreted as a diagnostic error; in view of our present knowledge of the subject such an occurrence is simply impossible.

The most diverse foods have been, more or less justly, charged with causing this disease; I give a few by way of illustration: The role of the starchy baby-foods, especially of such as contain desiccated milk, is indisputable (Still); cereals in general favor its development (Holst and Frölich), but Concetti² claims that this is true only when spoiled grain is used. The bad effects of homogenized, superheated, condensed, and desiccated milk have already been discussed under those headings; I merely cite the reports of Bourdillon, Bernheim-Karrer, Esser, and Hart. As to ordinary sterilized milk, opinions are far from unanimous. Finkelstein² is sure that it never is the sole cause of scurvy; Neumann says that only prolonged and excessive sterilization can render milk injurious; Schulz thinks that trouble may arise from the trace of silica that goes into solution when sterilization is carried out in glass vessels, and Plantenga considers sterilized milk perfectly safe if it is not kept over twenty-four hours. In this connection I may repeat that I have repeatedly observed scurvy in the children of physicians who had seen to thorough surgical sterilization of their babies' food. As to the various modifications of milk, Comby credits Gärtner's

fat-milk with producing scurvy, and Keller's maltsoup has also been regarded with warranted suspicion, although, according to Garrod's theory, the addition of potassium carbonate should act as a preventive.

Pasteurization is admittedly an imperfect method of sterilizing milk, and has only rarely been charged with producing this disease.

As to the four current theories of scurvy I must refer to a contribution by Hutchinson, who gives them in full; they cannot possibly be harmonized, and some modification of Garrod's view, to the effect that it is due to the deprivation of some necessary food ingredient, is most likely to prevail in the end, for the test of treatment very decidedly points in that direction.

Symptomatology and Diagnosis.—The following description will deal only with the infantile form of this disease, for which Morse⁷ has given a good outline of the symptomatology. The first sign is usually tenderness about the joints; next come gingivitis, and about the same time paresis (pseudoparalysis) of the extremities; later on we may witness hematuria and edema, but severe hemorrhages are rare because the affection is usually recognized and treated before it reaches this stage. Still and Snow mention orbital hemorrhage, with retrobulbar hematoma and consequent exophthalmos. We may go on to a more detailed description of some of these symptoms. The tenderness of the joints is so great that the baby screams out loudly if it be ever so gently moved; this is so characteristic that the diagnosis may often SCURVY 305

be made from this sign alone. Examination shows epiphyseal swelling, resembling that found in rickets; we cannot go astray if we remember that rickets is painless; what was formerly called acute rickets we now know to be scurvy. The frequently made diagnosis of rheumatism is quite unpardonable; articular rheumatism is very rare in infancy, and nearly or quite painless at that age. The diagnosis from other forms of acute arthritis, as well as osteomyelitis, is more difficult, but these latter affections are usually attended with considerable fever.

The inflamed gums are tender and swollen, bleeding at a slight touch; the diagnosis from infectious or aphthous gingivitis can usually be made at a glance; in the latter the presence of pus or isolated white spots is characteristic, and the swelling and hemorrhage are much less conspicuous. Subcutaneous and visceral hemorrhages occur so late in the disease that the symptoms, previously described, are sure to be present, otherwise the diagnosis from other forms, for instance, syphilitic hematuria, would be attended with some little difficulty. Should edema occur by itself it would of course be a very equivocal symptom; fortunately this is a most unlikely event. In my experience the diseases most resembling infantile scurvy, and most easily confounded with it, are infectious erythema (erythema multiforme) and the allied syndrome called Henoch's purpura, which may perhaps be due to an analogous nutritional disturbance, at least in some cases. Clinically, however, confusion rarely arises from these sources.

We are now in possession of an additional means of early diagnosis, namely, the x-rays. The Röntgen rays show the epiphyseal lesions before the appearance of clinical symptoms (Rehn, Hoffmann). The characteristic feature of scurvy is a dark shadow on the epiphyseal line, whereas in rickets we see an intensification of the normal light band at this site.

Treatment.—The treatment of infantile scurvy is exceedingly simple. The juice of the citrus fruits (orange, grape-fruit, lime, lemon) has been the sovereign remedy for centuries in the case of adult scurvy, and its effects have been quite as brilliant in early life, so much so as to render other remedies all but useless. Nevertheless, I may mention other antiscorbutics, namely, beef-juice, cabbage and its botanical allies (cress, turnips, mustard), and potatoes; none of these is quite as suitable for infants as orange-juice, given in doses of 2 or 3 teaspoonfuls before each feeding. It is also advisable to change to an antiscorbutic diet, with a basis of raw or moderately pasteurized milk, but this is not indispensable if the fruit-juice is given regularly and is well tolerated. It must be admitted that the action of fruit-juice is still very far from clear, and Garrod's theory, even if modified so as to apply to the organic salts of potassium, affords no real explanation. As a matter of fact our knowledge of scurvy has advanced but little since I4 wrote on the hemorrhagic diseases more than ten years ago, and our treatment is still actually quite empirical, though undoubtedly specific.

Orange-juice is also valuable as a convenient and

SCURVY 307

trustworthy prophylactic. It often happens that we are compelled to feed an infant on sterilized milk; since the unavoidable dangers, that lurk in raw milk, have become increasingly emphasized it is most gratifying to be enabled to give more or less sterilized food without incurring the risk of scurvy. This prophylactic treatment may be effectively accomplished by giving the infant one or two teaspoonfuls of orange-juice twice a week, a quantity quite sufficient to serve as a preventive. I have been doing this for some years in cases where artificial feeding with boiled milk was necessary. In view of a recent exposure I feel justified in a slight distrust of even certified milk, which I rarely give without preliminary heating, trusting to small doses of orange-juice to counteract any tendency to scurvy that may lurk in this imperfectly sterilized food. I never omit the orange-juice if I have occasion to resort to Keller's modification for any length of time, and have never seen scurvy arising under these conditions and precautions.

CHAPTER XIII.

THE FEEDING OF OLDER INFANTS.

Weaning.—Weaning, that is, removal from the breast, is physiological toward the end of the first year, but must sometimes be resorted to earlier, for various reasons. It is usually a perfectly safe procedure if the infant has attained the age of nine or ten months and has not presented any signs of the exudative diathesis, for normal infants of that age usually tolerate cow's milk well and often seem to gain more rapidly than before weaning. An exception should, however, be made if the infant attains the stated age during the summer months; in that case it is wiser to postpone weaning until the autumn, so as to avoid the risk involved in feeding with milk of doubtful purity.

Sometimes the supply of breast-milk begins to fail in the middle of the first year; when this happens we must, of course, go over to cow's milk, but we should always endeavor to interpolate a period of mixed feeding, giving the breast at eight-hour intervals, and two artificial feedings during the intermissions. In this manner, matters may often be tided over for several months until the normal weaning period.

Even at the age of nine months it is a good plan

to begin with mixed feeding, according to the above schedule, substituting the artificial food gradually, so as to extend the weaning period over a month if possible. Until the age of twelve months, as a general rule, the cow's milk should be diluted and sweetened by mixing eight ounces of barley water with thirty-two ounces of milk, adding an ounce of cane-sugar or milk-sugar, and dividing the whole into five eight-ounce portions.

The practice of prolonged breast-feeding, until the age of fifteen and even eighteen months, is met with in certain classes of our population, and is said to be usual in Japan. In some cases the infants appear to thrive and gain in weight on this feeding, but inquiry will almost always show that other food is also being given. Breast-milk alone is an insufficient food after the twelfth month, the supply gradually becoming inferior in quality even if the quantity remains ample; this practice of prolonged lactation should therefore, as a rule, be discouraged. Occasionally in the case of very poorly developed infants an exception may be made, but this eventuality is particularly rare, because the children of mothers whose lactation continues into the second year are apt to be rather above the average in general condition. In many cases the retardation of the infant's growth is actually caused by the gradual decline in the mother's milk-supply, and some of these infants improve with astonishing rapidity immediately after weaning.

Sometimes acute illness of the mother renders immediate weaning imperative; in this event we must

begin cautiously with a formula suitable to a somewhat earlier age, as the infant's tolerance for cow's milk is an unknown quantity. The process sometimes becomes extremely difficult, presenting in fact the entire problem of artificial feeding.

In weaning, the infant may at first refuse to take the rubber nipple; this may usually be overcome by a few hours of starvation, but some babies offer an obstinate resistance that can be overcome only by an exercise of the greatest patience. Equal difficulty is often afforded by the transition from the bottle to spoon feeding, but in these cases also the battle will usually be won by delaying or skipping a meal. Spoon-feeding should be begun before the fifteenth month, preferably about the turn of the year, because waiting longer is likely to make the change more difficult. The time-consuming labor of cleansing bottles and nipples should not be imposed upon the mother or nurse any longer than is necessary, and we can hardly begin the inculcation of proper habits of eating too early.

More objectionable even than excessively prolonged lactation is too early feeding with substances other than milk. This harmful practice is almost the rule in some countries, for example, Germany, where early feeding with pap is a fertile source of digestive and nutritional disorder (starch-atrophy). Starch-feeding, save in the very small amounts given in weak cereal decoctions, is quite unsuited to infants under the age of six months, and is best withheld for three months longer. There is no harm in waiting until

the end of the first year unless the child ceases to gain in weight on milk alone, and the perfect functionating of its digestive apparatus plainly shows that the trouble really lies with insufficiency of food. When this can be made out with certainty we may give a few ounces of well-cooked and strained cereal in milk, or an equal quantity of plain crackers, well broken up and soaked in milk, but no other kind of food should be added before the first year has elapsed.

Feeding During the Third Half-year.—After the twelfth month we may take the first steps in the direction of a mixed diet. This is facilitated, if we limit the total amount of milk to one quart per day, so that the child will desire other food; if we give large quantities of milk, as is sometimes done, the child may be satisfied with this alone, which is undesirable for two reasons. In the first place, milk has only one-fourth to one-half the caloric value of our common foods, there is therefore danger of insufficiency; secondly, it is not desirable to distend the stomach with unduly large quantities of fluid, which becomes inevitable if we try to feed an older infant on milk exclusively.

The number of meals should now be reduced to four per day; the five meals recommended by most authors are inconsistent with a sufficient interval, which should remain fixed at nearly four hours; I have found the hours of 7.30 and 11 A.M. and 3 and 6.30 P.M. the most convenient. Too frequent feeding is one of the causes of the anorexia that so often is a source of trouble and anxiety in very young children.

We may begin by giving the child crackers and rusks (zwieback), if those articles have not already been added to the dictary, as well as a well-cooked cereal once a day. I prefer the old-fashioned cereals, rice, farina, hominy, and oatmeal to the modern fancy cereal foods, which leave too little undigested residue and are therefore apt to favor constipation; oatmeal should be strained at first, but this is not necessary with the other cereals mentioned. The cereals may be somewhat sweetened, but an excess of cane-sugar should be avoided; too much strictness in this regard, however, is not justified, for there is no doubt that most persons who are fond of sweets can assimilate relatively large amounts of sugar; the desire for sweet things often indicates a physiological demand. A portion of the daily milk-supply should be used in cooking the cereal.

After a month or two we may add a soft-boiled or coddled egg to the daily dietary. Coddling consists in immersing the egg, for two or three minutes, in water that has been taken off the fire when the boiling-point has been reached; a coddled egg has a softer albumen than the ordinary boiled egg. Some authors recommend meat-juice at this period; we have seen that it contains very little nourishment, but the taste is usually relished, and a few teaspoonfuls can do no harm, and may act as an appetizer. Broths and soups are usually given at this age; plain meat broths are rather objectionable than otherwise, as they consist chiefly of a solution of salts and purin bodies; but cereal soups, such as rather thick rice

and barley broths, have some little food value. The total amount per day should not exceed four ounces; the common practice of filling up young children with large quantities of soup impairs the appetite for more nutritious food and tends to overload the stomach.

Feeding During the Fourth Half-year.—At the age of eighteen months, sometimes a little sooner or later, according to the development of the child's dentition, we may increase the variety of the food. Many infants now no longer take their full quart of milk when other food is given; some parents regard this seriously, but it is not an important matter if ample other nourishment is taken.

We may now begin to give scraped beef, chopped beef, and finely minced chicken, later on the finely cut inside of a lamb-chop; fat meats like pork and duck are unsuited to this age. Fresh fish is quite as good as fresh meat, and may be substituted for it once or twice weekly. There is a popular prejudice against giving meat before the age of two years, which has no physiological foundation whatever, for lean meat is one of the best means of giving the necessary protein, and should be included in one of the daily meals. Fat gravies, however, are absolutely prohibited. The egg ration may be continued, and the number may often be increased to two per day, one in the morning and one in the evening.

The use of crackers, rusks, and cereals should be continued, the soup ration on no account increased. We may now begin with bread, provided it is not too

fresh; a good plan is to reheat the bread of the preceding day in the oven, or to toast it; it may be buttered to suit the child's taste.

Fresh vegetables now are also in order, such as green peas, string beans, asparagus tips, spinach, carrots, and baked potatoes; all of these must be thoroughly mashed. There is no objection to stewed tomatoes if carefully strained. Fruits may also be given; in addition to the daily spoonful of orange-juice, applesauce and the pulp of stewed prunes are valuable means of regulating the bowels.

Tea, coffee, and cocoa, often given to very young children, are all very objectionable, and it should hardly be necessary to mention that alcohol in any form is a dangerous poison. At this age it becomes important to guard the child against the formation of the candy habit, which is often encouraged by thoughtless visitors and admirers. Before the age of two years the smallest amounts of candy or chocolate should be strictly banned.

We can now go over to the regular three meals per day, giving milk and crackers in the afternoon, to aid in spanning the long interval between luncheon and supper. A practical schedule is somewhat as follows:

8 A.M. One ounce of orange-juice.

Two ounces of cereal, with an ounce of cream and a teaspoonful of sugar.

Six ounces of milk.

Total about 250 calories.

11 A.M. or 12 M. Four ounces of thick vegetable or cereal soup.

Two ounces of meat or fish.

Two ounces of mashed potato or vegetable. One thick slice of bread with one teaspoonful of butter.

Total about 300 calories.

3 or 4 P.M. Two Graham or soda crackers. Six ounces of milk. Total about 200 calories.

6 or 7 p.m. One egg or two ounces of custard.
Slice of toast or a rusk.
Six ounces of milk.
Two ounces of cooked apples or prunes.
Total about 250 calories

This gives a total food value for the day of about 1000 calories, which is usually sufficient; the normal weight at this age being 11 to 12 kilograms (24 to 26 pounds), we have an allowance of 80 to 90 calories per kilo, which should be ample. It must, however, be remembered that we cannot lay down any hard and fast rules on this subject; some children will get on well with 70 calories, whereas others require at least 100, and complain of hunger when kept on the above diet.

Following C. H. Smith, I give the caloric values of various common articles of food, so that the reader may make calculations for himself:

Level tablespoon (approximately	y 1 ounce)	
Cane-sugar		60
Cream (16 per cent.)		50
Butter		115
Custard.		20
Fruit pulp		10
Cereals .		10
Mashed vegetables		15
Meat or fish		25
Milk		10
One cracker		30
" thick slice of bread.		75
" egg		75

These data are purposely given in round numbers, as any attempt at exactness is illusory; the above table will prove quite sufficient for all practical purposes.

Not more than six ounces of water (one goblet) should be taken at meals when milk is omitted; excessive thirst, when not due to illness, is usually a sign that the food is too salty or sweet, and calls for a corresponding correction in this regard.

Feeding Troubles.—During the second year, when the child begins to run about, great anxiety is often caused by a tendency of the weight to remain stationary; the thick layer of subcutaneous fat, acquired during the latter half of the first year by normal babies, is gradually lost. This condition is physiological; we should not expect a total gain of more than six or seven pounds in the course of the second year; so long as the child's spirits, sleep, and digestion are manifestly good there is no occasion for worry.

Real feeding difficulties are less common in the second year than in the first, but are by no means rare. A frequent occurrence is the persistent refusal of common and wholesome foods, usually certain vegetables. For this there are two possible reasons: In the first place many young children have predominantly carnivorous tastes; there is no great harm in an unusual craving for animal food, and, in the presence of this peculiarity, I do not hesitate to increase the ration of meat and eggs. Secondly, the insipid preparation of vegetables by the average American cook is largely responsible; the child simply has no relish for vegetables that taste like wet rags, and has trouble in overcoming this repugnance. In this event patience is necessary; we should see to tasty cooking of the vegetables, resorting to proper seasoning with salt, beef-juice, or meat-stock, and experiment with one variety at a time until the child learns to like it. We rarely have so much difficulty with other kinds of food; vegetables are evidently, to many children, an acquired taste.

Occasionally a child will refuse to eat eggs; I have seen such cases in families where there was a history of egg-idiosyncrasy; in such an event it is probably wisest to give way to the infant's prejudices.

Another common trouble is constipation, which sometimes is quite obstinate, especially in children who are suffering from rickets. It is desirable to correct this through dietetic measures, if at all possible, for there are many grave objections to the routine use of laxatives. The following expedients are often effective: To begin with I usually give unstrained oatmeal a prominent place in the cereal part of the daily regimen, but in rare cases an idiosyncrasy to

oats renders this method impracticable. Next, I order Graham crackers, which are keenly relished by most children, Graham bread, and the German ryebread, all of which are more laxative than ordinary white bread or crackers. Thirdly, I give mashed stewed fruit daily, and have found prunes decidedly more laxative than apples or other fruits, and often my main reliance. It is only when dietetic measures fail that we should resort to enemas or suppositories. If rickets is at the bottom of the trouble we must give phosphorus in malt, which I prefer to cod-liver oil, as being more digestible and palatable. Laxatives should be a last resort, and then the best drug is cascara sagrada, in a dosage that must be adapted to the individual. Medication with magnesia, calomel, senna, and, worst of all, rhubarb, is in my opinion highly objectionable.

In the fourth half-year it becomes necessary to give attention to the teeth, to proper habits of mastication, and the avoidance of bolting. Nervous children should be trained to attend strictly to business; the young gourmand who makes a rite of his meals is likely to do far better than the dreamer who allows his mind to wander from the food set before him. We must not forget that even at this early age the psychological study of the child becomes of great importance; some youngsters show signs of considerable intelligence long before the age of two years, so that patience and tactfulness are no unimportant factors in inculcating proper habits of eating. The bolting of badly masticated food is a specially serious

fault; it may lead to severe attacks of indigestion, with violent symptoms, as undigested masses of food travel, almost unaltered, through the entire digestive tract.

Some children give trouble as soon as attempts are made to vary their diet; these infants always present abnormalities in some other respects, with an almost invariable history of feeding difficulties during the first year. In these cases it often becomes necessary to return to formula-feeding for some weeks or months. I must, however, utter a word of caution against giving a low protein ration to these infants, as often advised; the trouble lies regularly with the cereal starches and never with the albuminous portion of the food. Carbohydrate overfeeding is indeed the usual error committed during the second year; many of these children get altogether too much cereal food, including bread and crackers, and if thereto is added an excess of potatoes or cane-sugar it is no wonder if we occasionally witness digestive upsets, often with fever, such as have already been described in carbohydrate and acid intoxications in younger infants. In fact the third half-year is by no means immune to the dangerous digestive disturbances of early infancy, and the mortality at this age in the summer months is not at all insignificant. This accounts for the popular impression as to the dangers of the second summer, which is well founded as regards such infants as have been fed at the breast during their first year.

This type of infant is best handled by imagining

it to be three or six months younger than its actual age, and feeding accordingly. It is safer to overlook a slight retardation of growth than to try to force the infant up to a regimen, of which the effective disposal is far beyond its feeble digestive powers.

BIBLIOGRAPHY.

Abt.—Arch. of Pediat., November, 1909. Ackermann.-Inauguraldiss., München, 1908. Adami.—British Med. Jour., January 24, 1914. Alexander and Bullowa.—Arch. of Pediat., January, 1910. Allaria.—1. Rivista di clin. pediat., June, 1912. 2. Ibid., July, 1912. Aron.—Biochem. Zeits., 1908, v. 12. Aronstamm.—Arch. f. Kinderh., 1903, v. 37. Aschenheim.—Monatss. f. Kinderh., 1913, v. 12. Auché et Campana.—Revue mens. des malad. des enf., 1906, v. 24. Avignaret et al.—Arch. de méd. des enfants, 1912.

Baginsky.—1. Lehrbuch der Kinderkrankh., 1905.

Arch. f. Kinderh., 1888, v. 9.

Berliner klin. Woch., 1894, No. 43.

Bahrdt.-Jahrb. f. Kinderh., 1910, v. 71.

Bahrdt u. Bamberg.—Zeits. f. Kinderh., 1911, v. 3.

Bahrdt u. Edelstein.-Zeits. f. Kinderh., 1911, v. 1.

Baker.—Arch. of Pediat., March, 1914.

Bamberg.—Zeits. f. Kinderh., 1913, v. 6.

Bauer.—New York Med. Jour., March 12, 1898.

Barlow.—Trans. Royal Med. and Chir. Soc., 1883.

Basch.—Münchener med. Woch., October 24, 1911.

Bauer.—1. Deutsche med. Woch., 1909, No. 38.

2. Monatss. f. Kinderh., 1911, v. 10.

Beardsley.—Reprinted in Arch. of Pediat., May, 1903.

Beattie and Lewis.—Jour. of Patholog. and Bacteriol., 1913, v. 18.

Beck.—1. Monatss. f. Kinderh., 1904, No. 5.

Ibid., 1912, v. 11.

Behring.—Deutsche med. Woch., 1903, No. 39.

Bendix.—1. Zeits. f. Kinderh., 1913, v. 6.

2. Deutsche Aerztezeitung, 1904.

Bendix u. Bergmann.—Monatss. f. Kinderh., 1912, v. 11.

Benfey.—Jahrb. f. Kinderh., 1912, v. 75.

Berend.—Monatss. f. Kinderh., 1913, v. 12.

Berg.—Biochem. Zeits., 1910, v. 30.

Bernheim-Karrer.—Correspond. f. schweizer Aerzte, 1907, No. 19. Bickel u. Roeder.—Berliner klin. Woch., January 3, 1910.

Biedert.—Virchow's Archiv., 1874, v. 60.

Birk.—1. Monatss. f. Kinderh., 1911, v. 10.

2. Ibid., 1910, v. 9, p. 595.

3. Ibid., June, 1908.

4. Berliner klin. Woch., July 3, 1911.

Monatss. f. Kinderh., 1910, v. 9, p. 279.

6. Ibid., 1910, v. 9, p. 140.

Bischoff.—Arch. f. Hygiene, 1903, v. 47.

Blühdorn.—Monatss. f. Kinderh., 1913, v. 12.

Boissonas.—Arch. de méd. des enf., 1908, v. 11.

Bordet.—Ann. de l'Inst. Pasteur, 1899, v. 13.

Bourdillon.—Rév. méd. de la Suisse romande, 1907, No. 20.

Bovaird.—Arch. of Pediat., June, 1909.

Bowditch and Bosworth.—American Jour. Dis. Child., March. 1914.

Brady.—1. Arch. of Pediat., June, 1910.

2. American Jour. Dis. Child., August, 1912.

Brennemann.—1. American Jour. Dis. Child., May, 1911.

2. Jour. Amer. Med. Assoc., February 22, 1913. Bruck.—Monatss. f. Kinderh., 1909, No. 8.

Brüning,—Arch. f. Kinderh., 1913, v. 60 and 61.

Brush.-Jour. Amer. Med. Assoc., June 20, 1903.

Budde.-Milk and its Treatment, 1896.

Budin et Planchon.-Rev. d'hyg. et méd. infant., 1904, v..3.

Calvary.—1. Ergeb. d. inn. Med. u. Kinderh., 1913, v. 10.

2. Zeits. f. Kinderh., 1911, v. 1.

Camerer u. Söldner.—Zeits. f. Biologie, 1898, v. 36.

Cardamatis.—Arch. de méd. des enf., 1904, No. 2.

Cassel.—Arch. f. Kinderh., 1912, v. 58.

Chapin.—1. New York Med. Jour., February 23, 1901.

2. Ibid., April 23, 1904.

3. Med. Record, February 18, 1905.

Ibid., May 28, 1910.
 American Med., March 4, 1905.

Chevalier.—La clinique infant., 1907, No. 20.

Chittenden and Griswold.—American Chem. Jour., 1882, v. 3

Chittenden and Mendel.—Osler's Mod. Med., v. 1.

Clarke.—Amer. Jour. Med. Sci., May and June, 1909.

Clock.—Jour. Amer. Med. Assoc., July 19, 1913.

Comby.—Archiv de méd. des enf., 1906, v. 10.

Concetti.—1. Rivista di clin. pediat., January, 1905.

2. Arch. f. Kinderh., 1909. v. 50.

Connstein.—Arch. f. Anat. u. Physiol., 1899.

Coolidge.—Arch. of Pediat., October, 1905. Coplans.—Lancet, October 19, 1907.

Cornet.—Nothnagel, spec. Pathol. u. Therap., v. 14.

Cowie and Hubbard.—Amer. Jour. Dis. Child., September, 1913.

Cramer.—1. Münch. med. Woch., November 13, 1900.

2. Monatss. f. Kinderh., January, 1908.

3. Arch, f. Kinderh., 1900, v. 32.

Cronheim, J. u. W.—Zeits. f. phys. u. diät. Therap., 1910, v. 14. Cronheim u. Müller.—Jahrb. f. Kinderh., 1903, v. 57. Curschmaun.—Deutsche Zeits. f. Nervenh., 1910, v. 39. Cybulski.—Monatss. f. Kinderh., November, 1906. Czerny.—1. Prager med. Woch., 1893, Nos. 41 and 42.

2. Monatss. f. Kinderh., 1905, No. 1.

3. Ibid., 1906, No. 11.

4. Ibid., 1907, No. 1.

Czerny u. Keller.—Des Kindes Ernährung.

Davidsohn.—Zeits. f. Kinderh., 1911, v. 2.

Davis.—Boston Med. and Surg. Jour., February 15, 1918.

Decherf.—Archiv. de méd. des enf., January, 1905.

de Jager.-Nederl. Tijds. v. Geneesk., 1895.

de Jong.-Nederl. Tijds. v. Geneesk., 1909, v. 1.

Deutsch.-Monatss. f. Kinderh., 1906, No. 7.

Dibbelt.—Beitr. z. path. Anat. u. allg. Pathol., 1910, v. 48.

Dombrowsky.-Arch. f. Hygiene, v. 50.

Donath.-Wiener med. Woch., 1910, No. 48.

Dubois u. Stolte.—Jahrb. f. Kinderh., 1913, v. 77.

Dunn.-1. Arch. of Pediat., April, 1907.

2. Ibid., June, 1905.

3. Boston Med. and Surg. Jour., November 18, 1909.

4. Jour. Amer. Med. Assoc., August 21, 1909.

Duval and Bassett.—Amer. Med., September 13, 1902.

Edsall.—Amer. Jour. Med. Sci., April, 1903.

Edsall and Fife.—New York Med. Jour., January 9, 1904.

Edsall and Miller.—Amer. Jour. Med. Sci., April, 1905.

Engel.—1. Arch. f. Kinderh., 1906, v. 43.

2. Deutsche med. Woch., June 26, 1913.

Engel u. Bode.—Zeits. f. physiol. Chemie, 1911, v. 74.

Epstein.-Prager med. Woch., 1896, No. 43.

Erving.—Arch. of Intern. Med., December, 1908.

Escherich.—1. Die Darmbakterien im Säuglingsalter, 1886.

2. Verh. d. Gesells. f. Kinderh., 1894.

3. Wiener klin. Woch., 1890, No. 40.

4. Münch. med. Woch., 1889, No. 13.

Esser.—Münch. med. Woch., 1908, No. 17. Eustache.—Ann. de méd. et chirurg. inf., 1904, No. 5.

Feer.—1. Jahrb. f. Kinderh., 1902, v. 56.

2. Korrespond. f. Schweizer Aerzte., 1904, No. 1.

3. Jahrb. f. Kinderh., 1913, v. 78.

Fife and Veeder.—Amer. Jour. Dis. Child., July, 1911. Finkelstein.—1. Lehrbuch d. Säuglingskrankheiten.

2. Therap. Monatsch., October, 1907.

3. Monatss. f. Kinderh., 1905, No. 5.

4. Jahrb. f. Kinderh., 1908, v. 68.

5. Deutsch, med. Woch., 1909, No. 5.

Finkelstein.—6. Jahrb. f. Kinderh., 1907, v. 65 and 66.

7. Amer. Jour. Dis. Child., May, 1912.

8. Berliner klin. Woch., October 9, 1911.

Mediz. Klinik, 1907.

Zeits. f. Kinderh., 1914, v. 8.

Finkelstein u. Meyer.-1. Berliner klin. Woch., June 20, 1910 2. Münch. med. Woch., February 14, 1911.

Flexner.—Johns Hopkins Hosp. Bull., 1900, v. 11.

Flexner and Holt.—Studies from the Rockefeller Inst., 1904.

Folin.—Jour. Amer. Med. Assoc., September 5, 1914.

Forest.—Arch. f. Kinderh., 1905, v. 42.

Francioni.—Rivista di clin. pediat., July, 1911.

Frank.—Monatss. f. Kinder., 1913, v. 12.

Frank u. Schittenhelm.—Zeits. f. physiol. Chem., 1911, v. 73.

Freudenberg u. Klocmann.—Jahrb. f. Kinderh., 1913, v. 78. Freund.—1. Biochem. Zeits., 1909, v. 16.

2. Jahrb. f. Kinderh., 1904, v. 59.

3. Zeits. f. physiol. Chemie, 1900, v. 29.

4. Monatss. f. Kinderh., 1908, No. 10.

Frew.—Lancet, November 4, 1911.

Friberger.—Münchener med. Woch., 1909, No. 38.

Friedjung u. Hecht.-Arch. f. Kinderh., 1903, v. 37.

Friedlander.—Jour. Amer. Med. Assoc., December 19, 1908.

Friedlander and Greenbaum.—Arch. of Pediat., September, 1912

Frost.—Arch. of Pediat., January, 1912.

Garrod.—Edinburgh Month, Jour. of Med., 1848.

Gärtner.-Verh. d. Ges. f. Kinderh., 1894.

Genersich.—Budapesti Orvosi Ujság, 1910.

Gewin.-Nederl. Tijds. v. Geneesk., 1912, v. 48.

Glaessner.—Wiener med. Woch., 1906, No. 40.

Glisson.—English Trans., 1651.

Goodall.—Arch. of Pediat., January, 1911.

Griffith.—New York Med. Jour., February 23, 1901.

Grósz.—Arch. f. Kinderh., 1905, v. 41.

Grulee.—1. Arch. of Pediat., June, 1904.

Amer. Jour. Dis. Child., September, 1911. Grulee and Buhlig.—Arch. of Pediat., October, 1911.

Haas.—Arch. of Pediat., March, 1914.

Hammer.—Bull. Iowa Agric: Exper. Sta., 1914, No. 15.

Hansemann.—1. Berliner klin, Woch., February 26, 1906

Ibid., 1899, No. 11.

Hart.—Jahrb. f. Kinderh., 1912, v. 76.

Hartje.—Jahrb. f. Kinderh., 1911, v. 73.

Hastings.—Jour. Amer. Med. Assoc., April 30, 1904.

Hecht.—Münch. med. Woch., 1908, No. 19.

Heim u. John.—Monatss. f. Kinderh., 1912, v. 11.

Zeits. f. Kinderh., 1911, v. 3.

Heiman,-1, Arch, of Pediat., August, 1910.

Heiman.-2. Ibid., December, 1911.

Helbich.-Monatss. f. Kinderh., 1911, v. 10.

Hemenway.-Jour. Amer. Med. Assoc., April 4, 1908.

Henoch.—Berliner klin. Woch., 1876, No. 18.

Herrman.-Arch. of Pediat., July, 1911.

Hess, A. F.-1. Amer. Jour. Dis. Child., March, 1913.

2. Ibid., May, 1912.

3. Ibid., October, 1912.

4. Deutsche med. Woch., 1912, No. 9.

5. Amer. Jour. Dis. Child., March, 1914.

Hess, J. H.—Amer. Jour. Dis. Child., November, 1911.

Heubner.—Jahrb. f. Kinderh., 1890, v. 32. 2. Therap, d. Gegenwart, 1906, No. 10.

Heubner, O. and W.—Jahrb. f. Kinderh., 1910, v. 72. Hichens.—British Jour. Child. Dis., September, 1913.

Hirschsprung.—1. Jahrb. f. Kinderh., 1888, v. 28.

2. Henoch's Festschrift, 1890.

Hochsinger.-Deutsche Klinik, v. 7.

Hoffmann.-Ziegler's Beiträge, 1905.

Holst and Frölich.—Jour. of Hygiene, October, 1907. Holt.—1. Diseases of Infancy and Childhood.

2. Arch. of Pediat., November, 1906.

3. Ibid., January, 1905.

4. Med. News, July, 1905.

5. Jour. Amer. Med. Assoc., June 27, 1914.

Holt and Levene.—Amer. Jour. Dis. Child., November, 1912. Hoobler.—1. Arch. of Pediat., November, 1910.

2. Amer. Jour. Dis. Child., August, 1911.

3. Arch. of Pediat., March, 1914.

Howland and Dana.—Amer. Jour. Dis. Child., July, 1913.

Hunaeus.—Biochem. Zeits., 1909, v. 22.

Hunziker.—New York Agric. Exper. Sta. Bull., No. 197, p. 61.

Hutchison.—Osler's Mod. Med., v. 1.

Ibrahim.—1. Zeits. f. physiol. Chem., 1910, v. 64.

2. Monatss. f. Kinderh., 1911, v. 10.

3. Die angeborene Pylorusstenose im Säuglingsalter, 1905.

4. Therap. Monatsch., 1908, p. 560.

5. Deutsche med. Woch., 1905, No. 23.

Jacobi.-1. New York State Jour. of Mcd., July, 1906.

2. Gerhardt's Handbuch d. Kinderh., 1876.

Jacobson.—Arch. de méd. des enf., 1909.

Jalland.—Med. Times and Gaz., 1873.

v. Jaksch.-Wiener klin. Woch., 1889, No. 22.

Jaschke.—1. Med. Klinik, August, 1908.

Monatss. f. Geburtsh. u. Gynäkol., 1909, v. 29.
 Jehle n. Pincherle.—Wiener klin. Woch., 1910, No. 3.
 Jordan and Mott.—Amer. Jour. Public Hygiene, 1910.

Kassowitz.-1. Zeits. f. klin. Med., 1883, v. 7.

2. Beiträge z. Kinderh., 1893.

3. Jahrb. f. Kinderh., 1884, v. 19.

Katzenellenbogen.—Monatss. f. Kinderh., 1911, v. 10.

Keller.—1. Arch. f. Kinderh., 1900, v. 29.

2. Monatss. f. Kinderh., 1910, v. 9.

3. Die Malzsuppe, 1904.

4. Berliner klin. Woch., September 3, 1906.

Kendall.—Boston Med. and Surg. Jour., November 20, 1913. Kendall and Smith.—Boston Med. and Surg. Jour., March 2, 1911. Kerley and Campbell.—New York Med. Jour., January 27, 1906. Kleinschmidt.—Monatss. f. Kinderb., 1912, v. 11.

Klotz.—1. Monatss. f. Kinderh., 1911, v. 10.

2. Ibid., 1909, No. 8.

3. Ibid., 1907, No. 5.

Knöpfelmacher.—Med. Klinik, 1908, No. 34.

Knox.—Jour. Amer. Med. Assoc., December 17, 1904.

Knox and Schorer.—Johns Hopkins Hosp. Rep., 1910, v. 15.

Knox and Tracy.—Amer. Jour. Dis. Child., June, 1914.

Kober.—Amer. Jour. Med. Sci., October, 1903. Koeppe.—1. Monatss. f. Kinderh., 1908, No. 6.

2. Deutsche med. Woch., June 17, 1909.

Kolff u. Noeggerath.—Jahrb. f. Kinderh., 1909, v. 70. Koplik.—1. Diseases of Infancy and Childhood, 1906.

2. New York Med. Jour., November 27, 1909.

3. Amer. Jour. Med. Sci., July, 1908.

4. Arch. of Pediat., March, 1907.

5. New York Med. Jour., January 31, 1891.

Krasnogorski.—Jahrb. f. Kinderh., 1910, v. 72.

Ladd.—1. Arch. of Pediat., October, 1904.

Ibid., March, 1908.

3. Ibid., May, 1912.
4. Boston Med, and Surg

4. Boston Med. and Surg. Jour., January 1, 1903.

5. Arch. of Pediat., June, 1910.

La Fétra and Howland.—Arch. of Pediat., March, 1904. Laisney.—Thèse de Paris, 1903.

Lamb.—Arch. of Pediat., June, 1908.

Langstein.—1. Jahrb. f. Kinderh., 1911, v. 74.

2. Ibid., 1906, v. 64.

3. Zeits. f. Kinderh., 1912, v. 5.

Langstein u. Meyer.-Jahrb. f. Kinderh., 1905, v. 61.

Langstein, Rott u. Edelstein.—Zeits. f. Kinderh., 1914, v. 8.

Laurentius.—Arch. f. Kinderh., 1911, v. 56. Leiner.—Wiener klin. Woch., 1904, No. 25.

Leo.—Berliner klin. Woch., 1888, No. 49.

Leopold.—1. Zeits. f. Kinderh., 1911, v. 1.

2. Arch. of Pediat., January, 1914.

3. Amer. Jour. Dis. Child., August, 1914.

Lewi.—See Holt. 1.

v. Liebig.—Suppe für Säuglinge, 1865.

Liepmann.—Berliner klin. Woch., 1912, No. 30.

Lissauer.-Jahrb. f. Kinderh., 1903, v. 58.

Litzenberg.—Amer. Jour. Dis. Child., December, 1912.

Lucas.—1. Arch. of Pediat., October, 1913.

Boston Med. and Surg. Jour., September 3, 1908.

Lust.-1. Monatss. f. Kinderh., 1912, v. 11.

- 2. Med. Klinik, 1912, No. 43.
- 3. Monatss. f. Kinderh., 1911, v. 10, p. 316.

4. Jahrb. f. Kinderh., 1913, v. 77.

Monatss. f. Kinderh., 1911, v. 10, p. 420.

6. Münch. med. Woch., 1913, No. 37.

McClure.—Amer. Jour. Dis. Child., January, 1914.

McCollum and Davis.—Jour. Biol. Chem., 1913, v. 15.

McLean.—Zeits. f. Kinderh., 1912, v. 4.

Malagodi.—Rivista clin. pediat., October, 1909.

Marfan.-1. Rev. des maladies de l'enf., 1895.

2. Arch. de méd. des enf., November, 1901.

Mayerhofer u. Prihram.-Monatss. f. Kinderh., 1910, v. 9.

Mazzeo.—La pediatria, 1905, No. 2.

Meeh.—Zeits. f. Biologie, 1879, v. 15.

Meigs.—Milk Analysis and Infant-feeding, 1885.

Meltzer and Auer.-Jour. Exp. Med., 1906.

Mendel.—1. Jour. Amer. Med. Assoc., September 5, 1914.

2. Amer. Journ. of Physiol., 1909, v. 24.

Menschikoff.—Monatss. f. Kinderh., 1910, v. 9.

Metchnikoff.—The Prolongation of Life, 1907.

Meyer, K.—Jahrb. f. Kinderh., 1913, v. 77.

Meyer, L. F.—1. Zeits. f. Kinderh., 1912, v. 5. 2. Monatss. f. Kinderh., 1906, No. 7.

Monatss. I. Kindern., 1906, No. 7.
 Berliner klin. Woch., 1907, No. 46.

4. Jahrb. f. Kinderh., 1910, v. 71.

Meyer and Leopold.—Arch. of Pediat., October, 1909.

Meyer u. Rietschel.-Berliner klin. Woch., December 14, 1908.

Michael.—Jour. Infect. Dis., January, 1905.

Michel.—L'obstétrique, 1896, p. 140.

Miller and Wilcox.—Lancet, 1907, No. 23.

Millon.—La pathologie infant., November 15, 1908.

Milner and Atwater.—Dietetics, Encycl. Brit., 11th ed.

Möller.-Medic. Jahrbücher, 1859.

Mori.—Monatss. f. Kinderh., May, 1905.

Moro.—1. Arch. f. Kinderh., 1906, v. 43.

2. Jahrb. f. Kinderh., 1902, v. 56.

3. Arch. f. Kinderh., 1904, v. 40.

4. Münchener med. Woch., 1908, No. 31.

Moro u. Kolb.-Monatss. f. Kinderh., 1910, v. 9.

Morse.—1. New Orleans Med. and Surg. Jour., August, 1910.

2. Amer. Jour. Med. Sci., March, 1904.

3. Amer. Jour. Obstet., May, 1910.

Morse—4. Amer. Jour. Dis. Child., November, 1911

Boston Med. and Surg. Jonr., May 28, 1903.

6. Arch. of Pediat., August, 1905.

7. Jour. Amer. Med. Assoc., April 14, 1906.

8. Amer. Jour. Dis. Child., May, 1911.

9. Amer. Jour. Med. Sci., November, 1912.

10. New York Med. Jour., March 8, 1913.

Morse and Bowditch.—Arch. of Pediat., December, 1906. Müller.—1. Jahrb. f. Kinderh., 1911, v. 73.

2. Therap. Monatsh., July, 1903.

Naunyn.—Nothnagel's spec. Pathol. u. Therap., v. 7, pt. 1 Neumann.—Berliner klin. Woch., 1905, No. 1. Niemann.—1. Jahrb. f. Kinderh., 1911, v. 74.

2. Jahrb. f. Kinderh., 1914, v. 79.

Nolf.-Le scalpel, 1910, No. 52.

Northrup.—Arch. of Pediat., January, 1905. Nothmann.—1. Arch. f. Kinderh., 1909, v. 51.

2. Monatss. f. Kinderh., 1909, No. 7.

3. Zeits. f. Kinderh., 1911, v. 1.

Ohno.—Philippine Jour. of Sci., 1906, v. 1. Oppenheimer.—Monatss. f. Kinderh., May, 1907. Oppler.—Monatss. f. Kinderh., 1903, No. 10. Orchard.—Arch. of Pediat., May, 1914.

Orgler.—Zeits. f. experim. Pathol. u. Therap., 1908, v. 5 2. Ergeb. d. inn. Med. u. Kinderh., 1908, v. 2.

Osborne and Mendel.—Jour. Biol. Chem., 1913, v. 16. Ott and Scott.—Therap. Gazette, October 15, 1911.

Park.-1. Jour. Med. Research, 1904, v. 11.

2. Arch. of Pediat., June, 1910.

Park and Holt.—Arch. of Pediat., December, 1903. Pedley.—British Med. Jour., October 20, 1906.

Peiser.—Deutsche med. Woch., September 12, 1912.

2. Monatss. f. Kinderh., 1912, v. 11.

Pelka.—Zeits, f. Kinderh., 1911, v. 2. Périer.—Ann. de méd. et chirurg. inf., 1903, Nos. 5 and 20.

Pfaundler.—1. Therap. der Gegenw., January, 1911.

2. Wiener klin. Woch., 1898, No. 45.

3. Münch. med. Woch., September 15, 1903. Pfersdorf u. Stolte.—Monatss. f. Kinderh., 1912, v. 11.

Philips.—1. Monatss. f. Kinder., April, 1907. 2. Ibid., November, 1906.

Pisek.—Arch. of Pediat., October, 1904.

Plantenga.—Arch. f. Kinderh., 1912, v. 58. Porter.—Amer. Jour. Dis. Child., August, 1913.

Potter.—Med. News, January 9, 1904.

Poynton.—Lancet, August 13, 1904.

Quincke.—Arch. f. exper. Pathol. u. Pharmakol., 1885, v. 19.

Raw.—British Med. Jour., August 18, 1906.

Reed and Ward.—Amer. Med., February 14, 1903.

Rehn.-Med. Klinik, 1907, No. 20.

Reinach.—Jahrb. f. Kinderh., 1904, v. 59.

Reuben.—Arch. of Pediat., March, 1914.

v. Reuss.—Zeits. f. Kinderh., 1912, v. 4.

Rietschel.—Monatss. f. Kinderh., 1910, v. 9.

Rivas.-Pediatrics, August, 1904.

Rommel.—Arch. f. Kinderh., 1903, v. 57.

Rosenow.—Jour. Infect. Dis., November, 1912.

Rosenstern.—1. Deutsche med. Woch., 1912, No. 39.

2. Ergeh. d. inn. Med. u. Kinderh, 1911, v. 7.

Rosenthal.—Nord. Tidskr. f. Terapi, 1906. Rotch.—1. Arch. of Pediat., August, 1904.

2. Jour. Amer. Med. Assoc., August 8, 1903.

3. Amer. Med., May 7, 1904.

4. Arch. of Pediat., February, 1893.

5. Text-book on Pediatrics, 1901.

Rotch and Kendall.—Amer. Jour. Dis. Child., July, 1911.

Rott.—Zeits. f. Kinderh., 1912, v. 5.

Ruhner u. Henbner.—Zeits. f. Biologie, 1899, v. 38.

Rudnik.-Wiener klin. Woch., 1906, No. 51.

Ruhräh.-1. Arch. of Pediat., July, 1909.

2. Amer. Jour. Med. Sci., April, 1914.

Salge.—1. Jahrb. f. Kinderh., 1912, v. 76.

2. Zeits. f. Kinderh., 1911, v. 2.

Samelson.—1. Zeits. f. Kinderh., 1911, v. 2. 2. Monatss. f. Kinderh., 1912, v. 11.

Schabad.—Zeits. f. klin. Med., 1910, v. 69.

2. Jahrb. f. Kinderh., 1911, v. 74.

3. Monatss. f. Kinderh., 1910, v. 9.

Schaps.—Berliner klin. Woch., May 13, 1907.

Schereschewsky.—Arch. of Pediat., December, 1913.

Schkarin.—Monatss. f. Kinderh., 1910, v. 9.

Schloss, E.—1. Jahrb. f. Kinderh., 1910, v. 71. 2. Ueher Säuglingsernährung, 1912.

Schloss, O. M.—Amer. Jour. Dis. Child., June, 1912.

Schloss and Crawford.—Amer. Jour. Dis. Child., March, 1911.

Schloss u. Frank.—Bioch. Zeits., 1914, v. 60.

Schlossmann.—1. Monatss. f. Kinderh., 1905, v. 4.

2. Arch. f. Kinderh., 1900, v. 30. Schlossmann u. Murschhauser.—Biochem. Zeits., 1910, v. 26.

Schlutz.—Amer. Jour. Dis. Child., February, 1912.

Schorer.—Amer. Jour. Dis. Child., April, 1912.

Schrack.—Jahrb. f. Kinderh., 1889, v. 29.

Schulz.—Münchener med. Woch., February 13, 1912.

Schwarz.—1. Amer. Jour. Dis. Child., September, 1911.

2. Arch. of Pediat., September, 1911.

Schwyzer.-Bioch. Zeits., 1914, v. 60.

Sedgwick.—1. Jahrb. f. Kinderh., 1906, v. 64.

2. Jour. Amer. Med. Assoc., October 1, 1910.

Sedgwick and Schlutz.—Amer. Jour. Dis. Child., October, 1911.

Seibert.—Med. Record, March, 1888.

Sharples and Darling.—Boston Med. and Surg. Jour., April 16, 1905.

Shelmerdine.—New York Med. Jour., March 14, 1908.

Shiga.—Centralbl. f. Bacteriol., 1898, v. 23.

Siegert.—1. Verh. d. Gesells. f. Kinderh., 1903.

2. Monatss. f. Kinderh., January, 1907, v. 5. Siegfried.—Zeits. f. physiol. Chem., 1896, v. 21 and 22.

Smith, C. H.—Arch. of Pediat., November, 1912.

Smith, R. M.—Boston Med. and Surg. Jour., November 20, 1913. Smith, Theobald.—Boston Med. and Surg. Jour., September 26,

1907.

Snow.—Arch. of Pediat., August, 1905.

Southworth.-1. Arch. of Pediat., March, 1910.

2. Ibid., February, 1907.

3. Ibid., September, 1912.

4. Ibid., February, 1905. Southworth and Schloss.—Arch. of Pediat., April, 1909.

Spolverini.—1. Riv. di clin. pediat., 1904, No. 2.

2. Arch. de méd. des enfants, March, 1904.

3. Revue d'hyg. et de méd. infant., 1908, No. 2.

Steele.—Arch. of Pediat., January, 1910.

Steinitz.—Monatss. f. Kinderh., 1902, No. 4.

Stern.—Arch. of Pediat., June, 1905.

Still.—British Med. Jour., July 28, 1906. Stolte.—1. Jahrb. f. Kinderh., 1911, v. 74.

2. Münch, med. Woch., December 17, 1912.

Stöltzner.—Jahrb. f. Kinderh., 1906, v. 63.

Stuhl.—Dentsche med. Woch., June 17, 1909. Székely.—Orvosi Hetilap., June 4, 1903.

or our results of the results of the

Talbot.—1. Amer. Jour. Dis. Child., March, 1911.

Boston Med. and Surg. Jour., June 11, 1908.
 Arch. of Pediat., December, 1909.

4. Amer. Jour. Dis. Chil., June, 1914.

Talbot and Hill.—Amer. Jour. Dis. Child., August, 1914.

Teixeira de Mattos.—Jahrb. f. Kinderh., 1902, v. 55. Thiemich.—1. Münch. med. Woch., 1910, No. 26.

2. Jahrb. f. Kinderh., 1896, v. 44.

Tobler u. Bogen.-Monatss. f. Kinderh., 1908, No. 1.

Tonney and Pillinger.—Jour. Amer. Med. Assoc., May 18, 1912.

Torrey and Rahe.—Jour. of Infect. Dis., v. 1.

Townsend.—Boston Med. and Surg. Jour., February 17, 1887.

Triboulet.—Bull. de la soc. pédiat. de Paris, 1909, p. 512.

Tugendreich.-Arch. f. Kinderh., 1906, v. 44.

Uffenheimer.—Ergeb. d. inn. Med. u. Kinderh., 1908, v. 2. Usuki.—Jahrb. f. Kinderh., 1910, v. 72.

Van Slyke.—Arch. of Pediat., July, 1905.

Variot.—1. Bull. de la Soc. pédiat. de Paris, 1913, p. 43.

2. British Med. Jour., May 14, 1904.

Vincent.—Amer. Jour. Dis. Child., February, 1914.

Vipond.—Montreal Med. Jour., April, 1910.

Vogt.-Monatss. f. Kinderh., January, 1910, v. 8.

Voix.—Thèse de Paris, 1903.

Volhard.-Münch. med. Woch., 1900, Nos. 5 and 6.

Voltz.—Biochem. Zeits., 1913, v. 52.

Wachenheim.—1. New York Med. Jour., September 9, 1905.

2. Amer. Jour. Med. Sci., April, 1905.

3. The Climatic Treatment of Children, 1907.

4. Med. News, January 16, 1904.

Wassermann.—Verh. d. 18. Kongr. f. inn. Med., 1900.

Weaver and Tunnicliff.—Jour. of Inf. Dis., January, 1905.

Weiland.—Berliner klin. Woch., July 13, 1908.

Weill et Mouriquond.—Jour. de méd. de Paris, June, 1912.

Weiss.—Volkmann's klin. Vorträge, 1880.

Wernstedt.-Monatss. f. Kinderh., August, 1905.

Westcott.—Arch. of Pediat., March, 1907.

Whitehead.—Amer. Jonr. of Physiol., 1909, v. 24.

Wilcox and Hill.—Amer. Jour. Dis. Child., April, 1913.

Wile.—Jour. Amer. Med. Assoc., March 16, 1912.

Williamson.—London and Edinburgh Month. Jour., 1841.

Winslow.—Boston Med. and Surg. Jour., December 14, 1911. Wollstein.—Jour. Med. Research, August, 1903.

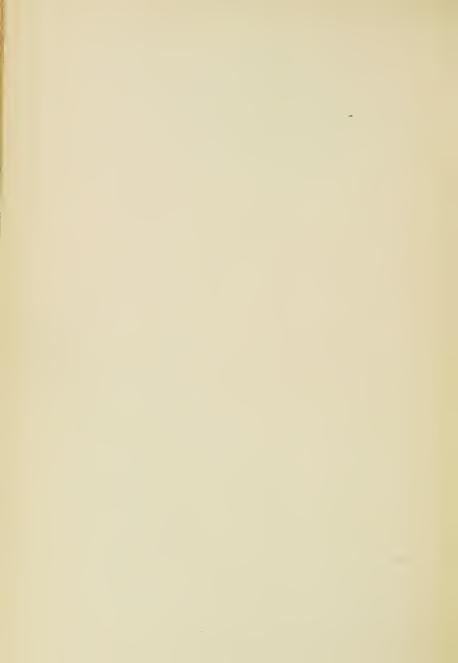
Wright.—Lancet, July 22, 1893.

Zahorsky.—Amer. Jour. Dis. Child., November, 1913.

Zaitschek.—Arch. f. d. ges. Physiologie, 1904, v. 54.

Zappert u. Jolles.—Berliner klin. Woch., 1903, No. 41. Zlocisti.—Berliner klin. Woch., February 1, 1904.

Zuckerkandl.—Wiener klin. Woch., 1905, No. 33.



INDEX.

A

ACETONE, 82, 224-5, 274 Acetonemia and Acetonuria, 223, 276Achondroplasia, 290 Acid, boric, 103 hydrochloric, 17, 20-1 Acidosis, 156, 224, 174-8, 287, Acids, fatty, 18, 22, 28, 33-4, 51, 82, 116, 166-70 in cow's milk, 82, 115-6, 166 - 70in human milk, 51 Adenoids, 270 Albumen-water, 177, 261 Albumin-cream-milk, 122-3 -milk, 118-22, 263-6 Albuminuria and nursing, 59-60 Albumose, 164 Alcohol, 54, 64, 164-5, 169-70, 260, 314 Alkalies Sodium). in acidosis, 282-3 addition to milk, 160-2 gastric secretion and, 18 Amino-acids, 21, 23 Ammonia, 23, 25, 37, 275 Amylase (see Diastase). Anaphylaxis, 281-2 to cow's milk, 108-9 to eggs, 177, 281 Anasarca (see Edema). Anemia in infants, 86, 272-4 nursing and, 60 Anorexia, 219

Antisepsis, internal, 21 Aphthæ, Bednar's, 67 Apples, 181, 314, 318 Aromatics as galactagogues, 54 Asparagus, 314 Asthma dyspepticum, 238 Atrophy from milk, 195, 213-41, 244, 263 from salts, 230 from starch, 147, 172, 223-5, 229, 276-7, 298, 310 Auto-intoxication, 240

В

Baby foods, 171-3 Bacillus cyanogenes, 76 dysentery, 249-50 gas-forming, 251 lactic acid, 32, 115, 166-9, 240tuberculosis, 89, 267-8 (see also Potassium, Bacteria, diarrhea and, 22, 234, 240, 248-51 in milk, 87-93, 97 ın top-milk, 125 relation to carbohydrates, 32, 263Bacterium coli, 251 Banana meal, 180 Barley, 34, 79, 148, 185, 190 Beans, 178 soy, 179 string, 314 Beef-extract, 176 juice, 174-5 Beer as galactagogue, 54, 64

Bile, 20 Bismuth, 260 Blood (see Anemia, Hemoglobin, Leukocytosis). Bones, 39 (see also Rickets). Bottle-feeding (see Feeding, artificial). Modification, Bottles (see home). Bread, 313-4, 316, 318-9 Breast, 53-7, 62, 66 of newborn, 54 Breast-feeding, 48-75 (see also Milk, human). capacity for, 54-7 contra-indications, 57-63 difficulties, 56, 61-2, 68-72 encouragement of, 71-2 prevalence of, 55-6 prolonged, 308-9 rules for, 64-8 Breast-pump, 53, 56-8, 237 Broths, 175-6, 312-3 Butter, 316 Buttermilk, 115-8, 168, 233, 235, 262

C

Caffein, 260 Calcium, administration of 283, 293-4, 301 in cow's milk, 77 in human milk, 50-1 metabolism, 32, 35, 37-39, 83, 285-7, 297 soaps, 28, 33, 37, 83 Calomel, 70, 259, 318 Caloric feeding, 46-7 requirement, 35, 43-7 values, 44, 314-6 Camphor, 260 Carbohydrates, metabolism of, 21, 30-5, 42, 83-4, 145-9, 155-8, 222-5 (see also Dextrin, Malt, Starch, Sugar). Carbon dioxid, 40 Carcinoma and nursing, 60 Cardiospasm, 215, 296 Carrots, 179-80, 273, 314

Cascara sagrada, 64, 318 Casein in cow's milk, 78-9 curds in feces, 28, 80-1 in human milk, 50 Casein-fat-milk, 141-2 Castor oil, 69, 70, 195, 259, 282 Cereals, 34, 147-8, 312-3, 316, 319 (see also Barley, Oatmeal, Rice, Starch, Wheatflour). Chlorids in cow's milk, 77 in human milk, 50 Cholera infantum, 101, 239 (see also Dysentery). Cocoa, 314 Cod-liver oil, 27, 294-5, 318 Coffee, 65, 314 Colic, 68-9, 193, 234 Colostrum, 24, 48-9, 57 Constipation, 21, 33, 69, 96, 190-3, 317-8 in nursing mothers, 64 Corpus luteum extract as galactagogue, 53 Cotton seed as galactagogue, 53 Cows, breeds of, 76-7, 125 Crackers, 311-4, 319 Cream, 316 (see also Top-milk). Creatinin, 23, 25 Curds, casein, 28, 80-1 fat, 28, 80 Custard, 316 Cyanosis, 236 Cystitis, 270

D

Death-rate, 91-2, 245-8
Decomposition, 235-8 (see also Atrophy).
Dentition, 68, 217-8, 270, 289, 318
Dermatoses, metabolic, 278-82
Dextrin, metabolism of, 31-3, 145-6, 155-8
Dextrose (see Glucose).
Diarrhea, 22, 33, 69, 194-6, 238 fat, 29, 196, 236
from frozen milk, 101
summer (see Dysentery).

Diastase, 17, 20, 31 in human milk, 52 Diet of nursing mother, 64-5 of older infants, 311-6 Digestion, 17-22 disorders of, 22, 206-66 Dispensaries, 106-7 Drugs in milk, 65 Duodenal digestion, 20 tube, 20, 212 Dysentery, 239, 243-66 bacteriology, 248-51 etiology, 91, 243-51 pathology, 251-2 prognosis, 254-5 prophylaxis, 255-8 serotherapy, 260-1 symptoms, 252-4 treatment, 258-66 Dyspepsia, 233-5, 244

E

Eclampsia, nursing and, 59 Eczema, infantile, 269, 278-80 of nipples, 57 Edema, 229-30, 236-7, 253, 304-Eggs, 27, 176-8, 261, 281, 312-3, 316-7 Enemas, 192, 318 Energy output, 43 quotient, 44 storage of, 44 Enterokinase, 20 Epilepsy, nursing and, 60 Equilibrium, disturbed, 231-3, 244Erepsin, 21 Extractives, 164, 174-5 (see also Purin bodies). Exudative diathesis, 71, 267-72, 279-80, 290

F

FARINA, 312 Fats, caloric value of, 35, 44-5 Gases, metabolism of, 40

Fats, chemical tests for, 29 in cow's milk, 76 in human milk, 50-1 metabolism of, 18, 25, 27-30, 34, 38, 42, 82-3, 221-4, 285-6Feces (see Stools). Feeding, artificial, 184-205 (see also Milk, cow's). caloric, 159-60 duration, 66 in fourth half-year, 313-6 in second half-year, 198-9 interval, 19, 65-6, 68-9, 126 in third half-year, 311-3 mixed, 199-201, 308-9 night, 65-6, 69, 152 percentage, 128-39, tables, 129, 139, 152 Fermentation, alcoholic, 169-70intestinal, 192, 234 lactic acid, 32, 166-70 Ferments, administration of. 162-70 in cow's milk, 74 in human milk, 52 Fever, contra-indication to nursing, 61 inanition, 42 in dysentery, 253 salt, 225-8 sugar, 110, 117, 144, 226, 262-3 thermic (see Heat-stroke). water in, 183 Finkelstein's theory, 231-41 Flies, 256 Flour (see Starch). Foot and mouth disease, 90 Formaldehyde, 65, 102 Fruits (see also Apples, Oranges, Pineapples, Prunes), 316

G

GALACTAGOGUES, 53-4 Galactose, 31

Gastric juice, 17 (see also stomach) Glucose, 31 Glycerophosphates, 38 Glycogenic function, 31, 239 Glycolytic ferments, 165-70 (see also Diastase, Invertase, Lactase, Maltase). Goitre, exophthalmic, and nursing, 60 Growth, 40, 198 Gruels, 34, 147-9 Gums, lancing of, 68

H

Heart disease and nursing, 60 Heat, body and, 34, 44 death rate and, 95, 244-7 intestinal infection, and, 91, 244 - 8stroke, and, 44, 247-8 water metabolism, and, 40, 182 Hemoglobin, 38, 86, 272 Hemp-soup, 180 Hiccough (see Singultus). Hirschsprung's disease Megacolon). Hominy, 312 Hospitalism, 53 Hospitals, floating and seaside, 256,258Humidity, 44, 91 Hydrogen peroxid, 74, 102 sulfid, 32 Hydrolytic ferment in human milk, 52 Hygiene, instruction in, 256-7 Hypertony, 223, 295 (see also Muscular Rigidity). Hypophosphites, 38 Hysteria and nursing, 60

Ι

Icterus neonatorum, 20 Ileocolitis (see Dysentery).

Imperial Granum, 173 Incubator, 44, 204-5 Indigestion (see Dyspepsia). Indol, 32 Infection, intestinal (see Intestinal Infection). Infectious diseases, acute, and nursing, 61 Infusion, saline, 228, 260 Insanity and nursing, 60 Intestinal fermentation, 192, 234infection, 88-92, 222, 234, 243 - 66Intestine, functions of, 18, 21-2, 27, 31-2 length, 21 Intoxication, 100, 117, 226, 238-42, 262-3, 298 Invertase, 21, 31 Iron in cow's milk, 37, 77 in human milk, 37, 50-1 medication, 273 metabolism, 37-8, 85-6, 272

J

(see JUNKET, 118

K

Kefir, 169-70 Kumyss, 169-70

L

Lactalbumin (see Proteins, Whey).
Lactase, 21, 31
Lactoglobulin (see Proteins, Whey).
Lactose (see Sugar, Milk).
Lactosuria, 238
Laryngospasm, 296, 299
Lavage, 213, 259
Laxatives, 64, 193, 317-8
Lecithin, 38, 51-2, 96, 176 (see also Phosphorus).

Legumes, 178-9 Lentils, 178 Leukocytes and uric acid, 26 Leukocytosis, 26, 238, 240 Levulose (see Glucose). Lime (see Calcium). Lime-water, 161, 283 Lipase, 17-8, 20, 27 in human milk, 52 Liver, 31, 289 Lymphatism, 268, 271, 279 Lymph-nodes, 289

M

Magnesium, administration of, 193, 301, 318 metabolism of, 28, 35, 38 Malt, 233, 295, 318 (see also Dextrin, Maltose). as galactagogue, 54 Maltase, 17, 21, 31 Maltose, 31, 33, 145, 155-8 Malt-soup, 155, 235 Mastication, 318 Mastitis (see Breast). Matzoon, 166-8 Meat, 313, 316 Meconium, 30, 42 Megacolon, 214-5 Mehlnährschaden (see Atrophy from starch). Menstruation and nursing, 61 Metabolism, disorders of, 267-307 of carbohydrates, 21, 30-5, 42, 145, 222-5 of fats, 27-30, 42, 82-3, 221-4 of gases, 40 of proteins, 22-7, 42, 82, 220-1, 224-5 of salts, 35-9, 83-6, 225-30, 237, 272of water, 39-40, 181-3, 228-30 Milchnährschaden (see Atrophy from milk). Milk, acidified, 116, 166-70, ass's, 110

Milk, cow's, analysis of, 77-8 bacteria in, 87-94 bactericidal power of, 86-7 blue, 76 caloric value of, 76-7, 316 certified, 93-4, 307 composition of, 76-7 condensed, 100-1, 303 desiccated, 101, 303 dilution of, 18, 111-2 ferments in, 74, 96 frozen, 101-2 homogenized, 170-1, 303 idiosyncrasy, 108-9 malted, 155-8, 173, 304 modified, 111-73 (see also Modification of milk). pasteurized, 94, 97-100, 249, 304 peptonized, 163-4 preserved, 102-3 raw, 81, 94 regulations for, 93-6 skimmed, 114 sterilized, 91, 96-7, 300 supply of, 92, 255 whole, 107-8 goat's, 110 human, bactericidal power of, caloric value of, 50 composition of, 50-3 drugs and, 65 examination of, 63-4 ferments in, 52, 74 quality of, 61-2, 71 quantity of, 54, 61-2, 69-70 200preservation of, 74-5 secretion of, 53-5laboratories, 130-2 mare's, 110 stations, 92, 103-7, 257-8 Modification of milk, 111-173 Backhaus', 164 Baner's formula, 134-5 Biedert's, 126-7 Feer's, 122-3 Finkelstein's, 118-22 (see also Albumin-milk)

Modification of milk, Gärtner's, 139-40, 303 Heim and John's, 141-2 home, 132-3, 137-8, 189-90 Horlick's, 173 Jacobi's, 149-54, 185-8 Keller's, 155-8, 235, 304 Liebig's, 155 Meigs', 127-8 Nestlé's, 172, 233, 298 Niemann's, 141 Pfaundler's, 154-5 Rotch's, 128-39 Schloss', 143 Székely's, 140-1 Mouth, care of, 58; 67 Mucus in feces, 29, 252 Muscular rigidity and spasm, 298 296, 147, (see Hypertony).

N

Nephritis in dysentery, 240, 253nursing and, 59-60 Neuroses, digestive, 217-9 Nervous diseases and nursing, 60 system and phosphorus, 39 Newborn, metabolism of, 41-2, 45 Nipples (see Breast). Nitrogen (see also Proteins). intestinal, 23, 25 metabolism of, 22-7, 42Nuclein, 26, 96 Nursing (see Breast feeding). Nystagmus, 296, 300

0

Oatmeal, 34, 148, 191, 281, 293, 312, 317 Opium, 259 Oranges, 181, 306-7, 314 Osmosis, 36, 226 Overfeeding, 68-9, 194 Oxydase in human milk, 52 Oxygen, 40

P

Pacifiers, 67-8 Pancreas, 20 Pancreatic extract, addition to milk of, 163-4 Parathyreoid glands, 297-8 Pasteurization, 94, 97-100, 249 Peaches, 181 Peas, 178, 314 Pepsin, 17 addition to milk of, 163 Peptones (see Albumose). Percentage feeding, 128-39 tables, 129, 139 Peristalsis (see Intestine, Stomach). Perspiration, 40, 182 Pharyngospasm, 215, 296 Phosphorus, administration of, 293-5, 318 in cow's milk, 77 in human milk, 50-2 metabolism, 38-9, 85, 285-7 Phytin, 38 (see also Phosphorus). Pineal gland extract as galactagogue, 53 Pineapples, 181 Pituitary gland extract as galactogogue, 53 Pneumococcus, milk-borne, 94 Potassium, administration of, 155, 277, 298 in cow's milk, 77 in human milk, 50 metabolism, 35-6, 84 sulfocyanid, 17 toxicity of, 36 Potatoes, 179, 306, 314, 319 Pregnancy, nursing, and, 61 Premature infants, 201-205 Premiums for nursing, 72 Proteins, caloric value of, 44-5 in colostrum, 24, 48-9 in cow's milk, 76, 78-82 in human milk, 50

Proteins, metabolism of, 18, 21-7, 42, 82, 220-1, 224-5 vegetable, 24-5, 156, 178, 224 whey, 50, 78, 81
Proteolytic ferments (see also Pepsin, Rennin, Trypsin). addition to milk of, 163-5 in human milk, 52
Purin bodies, 26, 175, 312 (see also Extractives).
Putrefaction, intestinal, 32
Pylorus, 18
spasm of, 208-14, 296

R

stenosis of, 206-14

Recreation piers, 256, 258-9 Rennet (rennin), 17-8, 163 Respiration, 40, 238 Cheyne-Stokes, 236 Rhubarb, 70, 193, 318 Rice, 312 Rickets, 96, 123, 157, 172-3, 193, 271, 274, 284-302, 317 Röntgen rays, 306 Rumination, 216

S

Saliva, 17, 31 Salt-fever, 225-8, 241-2 Salts, in cow's milk, 35, 76-7, 84-6in human milk, 35, 50-2 metabolism of, 34-9, 225-30, 237Salznährschaden (see Atrophy from salts). Scarlet-fever, milk-borne, 90 Scrofulosis, 267-8 Scurvy, 96-7, 100-1, 123, 157, 171-3, 181, 302-7 Seaside hospitals, 256, 258 Senna, 318 Silica, 303 Singultus, 216 Skatol, 32

Skim milk, 114 Soaps, 28-9, 32, 37, 83, 196-8, $2\bar{3}2$ Sodium benzoate, 103 bicarbonate, 161, 277, 283 chlorid, 18, 35-6, 50, 77, 84, 225-30 citrate, 161-2, 283 fluorid, 103 Somatose (see Albumose). Soups (see Broths). Spasm, nodding, 296 Spasmophilia, 295-302 Spinach, 273, 314 Spitting, 193-4 Starch, metabolism, 31, 33-4, 222-4(see 146-9, alsoAtrophy from starch). Starvation, 18, 223-5, 236, 238, 275, 277, 298 treatment by, 69, 237, 261-2, 280Sterilization, 91, 96-7, 300 Stomach, 17-18, 21 Stomatitis, 58 Stools, blood in, 236, 253-4 calcium in, 28, 37 curds in, 28 fats in, 27-9, 196, 236 ferments in, 30 green, 29-30, 69 hunger, 70, 207 in artificial feeding, 28-30, 197mucus in, 29, 252 normal, 27-30 protein loss in, 23 pus in, 253-4 soap, 28-9, 37, 196-8, 232 Streptococcus infection, milkborne, 90-1, 94, 251 Sucking, act of, 17 Sugar and gastric secretion, 18 intoxication, 100, 117, 144, 222, 226, 262-3 metabolism of, 30-5, 145, 222Sugar, cane, caloric value, 316 metabolism, 31, 145 milk-, in cow's milk, 76 in human milk, 50-1

Sugar, milk-, metabolism, 31-2, | Urine, 23, 26-7, 38 83-4, 145Sulfur, metabolism, 39 Suppositories, 192, 318 Syphilis and nursing, 59, 73

T

Tannin, 260 Tea, 65, 314 Teething (see Dentition). Teething-rings, 67 Temperature of cities, 244-7 indifferent, 44 Tenements, 248, 256 Tetany (see Spasmophilia). Thymus extract as galactagogue, 53 Thyreoid extract and protein metabolism, 25 Tomatoes, 314 Tongue-tie, 68 Top-milk, 123-41 Trypsin, 20 addition to milk, 163 Tuberculosis, 267-8 milk-borne, 90-1 nursing and, 58, 73 Twins, 50, 53 Typhoid fever, milk-borne, 88-9

U

Underfeeding (see Starvation). Urea, 23 Uremia and nursing, 59 Uric acid, 26-7

Urticaria, 279-82

v

Vacuum bottle, 98-9 Vegetables, 178-80, 314, 316-7 Vernix caseosa, 42 Vomiting, 21, 193-4 neurotic, 218-9 periodic, 276

W

WATER, 181-3, 316 matabolism, 36, 39-40, 228-30Weaning, 308-11 Weight, 26, 40-2, 316 Wet-nursing, 72-4 Wheat-flour, 34, 148 Whey, 112-5, 262 proteins, 50, 78, 81 Whey-modified milk, 143 Whooping cough, 300 Worms, intestinal, 281-2

Y

Yeast, 169-70 Yoghourt (see Matzoon).

Z

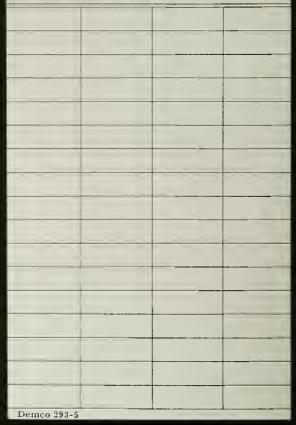
Zoolak (see Matzoon).







Date Due





RJ216 915W

